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CUTANEOUS LEISHMANIASIS IN SOUTH AUSTRALIA.

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THE migrants who come to Australia bring with them special medical problems, and Last (1960) has recently described some of these. One which he did not mention, and which has received very little attention, is cutaneous leishmaniasis. Experience in South Australia suggests that this is not uncommon in Italian migrants. There are parts of Southern Italy where the disease is established, and it is endemic in the countries bordering the eastern Mediterranean Sea, and in Pakistan and northern India. Thus migrants and students from a number of countries could bring the disease to Australia. It is unlikely that the disease would become endemic in South Australia, but from all points of view, including the prestige of Australian medicine in the eyes of the migrant population, it is desirable that the diagnosis should be made as soon as possible. The clinical features are reasonably distinctive, but the diagnosis can be proved only by demonstrating the parasite microscopically. The paucity of reports in the Australian literature may mislead clinicians and pathologists about its frequency, and this paper was prepared with the purpose of making the disease better known.

In the years 1955 to 1958 I saw five examples of cutaneous leishmaniasis in approximately 4000 skin specimens examined at the Institute of Medical and Veterinary Science, Adelaide. I was also able to see four of the patients, through the courtesy of the doctors attending them, and brief clinical details are given below. All five patients were Italian, and another probable case has been described to me by Dr. John Last, who has, however, been unable to trace the patient, an uncooperative Italian, to confirm the diagnosis by biopsy.

The first case of this series has already been reported by Donald (1958), who gives a good account of the epidemiology and treatment of the condition. I will confine my attention to the clinical and histopathological features of the disease.

Case I.

An Italian woman, aged 20 years, who had come to Australia five years before, developed a small fleshy papule on her nose twelve months before Dr. Gordon Donald asked me to perform a biopsy. Her previous treatment had consisted of a course of radiotherapy of tumour dosage (given by a radiotherapist) without benefit, and an oral course of mepacrine and chloroquine given by Dr. Donald, which arrested the development of

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the lesion for a few months. Examination showed an infiltrated nodule 2 cm. in diameter with a central crusted ulcer. At the periphery there were several tiny papules, and vitropressure showed a yellowish translucent infiltrate not unlike that seen in lupus vulgaris. Histological examination revealed a chronic inflammatory granuloma, with numerous parasites resembling *Leishmania tropica* in the cytoplasm of the macrophages.

Case II.

An Italian woman, aged 23 years, was examined by Dr. A. J. Hakendorf at his skin out-patient department at the Royal Adelaide Hospital, with a history of a swelling on the left side of her forehead present for

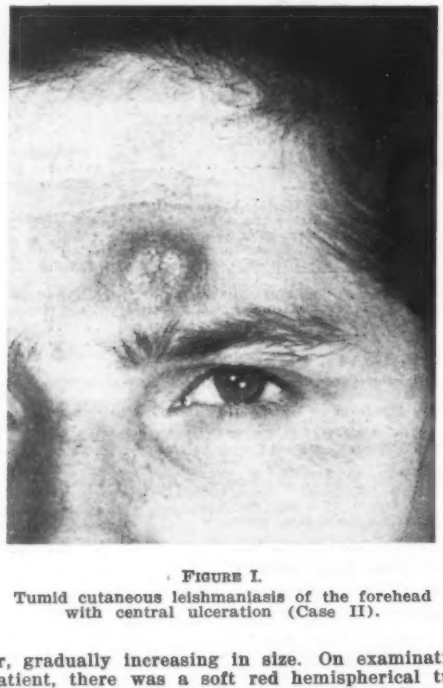

FIGURE I.
Tumid cutaneous leishmaniasis of the forehead with central ulceration (Case II).

a year, gradually increasing in size. On examination of the patient, there was a soft red hemispherical tumour 2·5 cm. in diameter, over the inner end of the left eyebrow. It was rather tender to pressure and not fluctuant, and there was no crusting or ulceration. Vitropressure disclosed a brownish-yellow infiltrate. A biopsy was performed, and central ulceration occurred afterwards. The appearance at this stage is shown in Figure I. The section showed the feature of a dense chronic inflammatory granuloma, with small numbers of *L. tropica* in the deeper parts.

Case III.

A male Italian, aged 35 years, was examined by Mr. Mervyn Smith at his surgical out-patient clinic at the Royal Adelaide Hospital, with an ulcer on the back of the lower part of his left leg. He stated that this had been present for four weeks. There was no history of any lesion preceding the ulcer. The man had been in Australia for two years. Examination showed a granulating ulcer 3 cm. in diameter, with a raised dusky red margin about 1 cm. in width (Figure II). It was situated on the posterior aspect of the lower part of the left leg, 8 cm. above the ankle joint. Vitropressure of the margin revealed yellowish infiltration with some miliary satellite areas further out. A Wassermann test performed on the serum had produced a negative result. Biopsy proved the diagnosis to be cutaneous leishmaniasis. A very good preparation showing the parasites was made by smearing on a slide scrapings from the surface of the ulcer.

Case IV.

An Italian woman, aged 30 years, was examined at the request of Dr. W. C. T. Upton, who considered that she was suffering from cutaneous leishmaniasis, and wished a biopsy to be performed. She had come to Australia from Naples two years previously, and for one year she had noticed a pimple on the left cheek which had gradually been enlarging. Examination showed this to be an elliptical area of dusky inflammation, with a yellowish infiltration of the peripheral part and scarring of the central area. A biopsy was done, and scrapings were made into smears and stained. No protozoa were seen in the smears. The histological appearance was that of a tuberculoid granuloma. Islands of epithelioid cells were bounded by lymphocytes, and some multinucleated giant cells were present in the infiltrate. A few parasites resembling *L. tropica* were seen in the deeper part of the granuloma. The appearance otherwise could easily have been taken for lupus vulgaris.

Case V.

A specimen of skin, lenticular in shape, measuring 2·2 cm. by 1·0 cm., was received at the Institute of Medical and Veterinary Science. It was stated to have come from the arm of a man with an Italian name, and

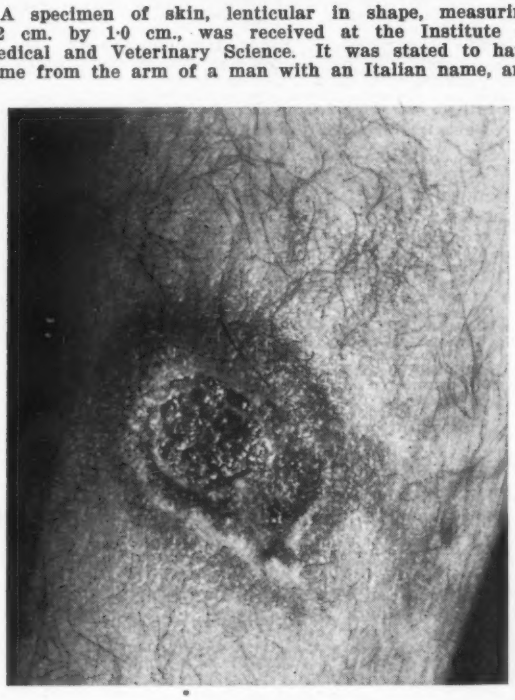

FIGURE II.
Ulcerated lesion of the lower part of the leg. The base of the ulcer is moist and granular. The edge is undermined and the margin is raised and infiltrated (Case III).

to be the excised portion of a tumour. Further clinical details could not be obtained. The centre of the specimen was occupied by a slightly elevated brownish area 0·5 cm. in diameter. Microscopic examination showed this to be a chronic granuloma containing large numbers of protozoa within macrophages.

Discussion.

Perhaps the most remarkable feature of cutaneous leishmaniasis is the extremely long latent period which may occur between the infection and the development of the sore. Donald has remarked that the four-year period in his case, though uncommon, is not exceptional. Unless clinicians are aware of this prolonged latent interval, the disease may be dismissed from the differential diagnosis of a chronic granuloma on the grounds that the patient has lived in Australia for several years.

The clinical appearance of the lesion is sufficiently characteristic in most cases to suggest the correct diagnosis. It occurs on areas exposed to biting insects, and starts as a small papule which enlarges, often quite rapidly, to become an inflamed nodule up to several centimetres in diameter. The colour at this stage is a rather dusky red, and if this is blanched by pressure with glass, a yellowish stain is revealed as in lupus vulgaris. Sooner or later the centre breaks down, leaving a crusted indolent ulcer with a raised margin. Satellite papules may develop at the periphery. There may be several such lesions on the exposed skin. The disease usually runs a course of many months. There is a strong tendency to spontaneous healing, which leaves a depressed atrophic scar.

In the earlier stages the condition may be mistaken for an "indolent boil" or for ecthyma. Later the possibility of tertiary syphilis, lupus vulgaris or a tuberculous gumma must be considered. The lesion is not likely to be confused with a neoplasm, because it lacks induration. In the tumid stage the soft boggy inflammation distinguishes it from the non-infective granulomas. However, the final diagnosis can be made only by demonstrating the specific organism, either in scrapings from the granulomatous tissue or in sections of a biopsy specimen. The organism can be cultured from the scrapings, a procedure especially valuable when few parasites are to be found by direct examination.

Apart from the presence of the protozoa in the tissue, the histological findings are not specific. However, some features are very suggestive and should stimulate a careful search for the organisms. At all stages there is an inflammatory infiltrate in the dermis, which is composed of a mixture of histiocytes and lymphocytes with a smaller number of plasma cells and sometimes polymorphonuclear leucocytes. In the earlier stages the histiocytes predominate, and many will contain a number of *L. tropica* organisms in their cytoplasm. Lymphocytes are scattered haphazardly between these cells and at the margins of the infiltrate. The granuloma extends up to the epidermis, which tends to be flattened and lacks the perivascular concentration which syphilitic lesions, for instance, show (Figure III).[1] The normal architecture of the dermal connective tissue is obscured, and the hair follicles and sweat glands show more or less damage. When ulceration occurs, the floor of the ulcer may show evidence of bacterial infection, and the epidermis at the margins may be hyperplastic to a degree suggesting epitheliomatous change. The later lesions become tuberculoid, with islands of epithelioid cells surrounded by rings of lymphocytes. Giant cells may be seen, but there is little tendency to central necrosis of these foci. At all stages the vessels are dilated and œdema is apparent.

The organisms are found in macrophages in areas where these cells are plentiful, but they are scanty or absent where the histiocytes have become grouped into a tuberculoid formation. Even with a low-power (16 mm.) objective, their presence can be suspected because of a faint basophilic stippling of the macrophage cytoplasm. They can be identified without difficulty with an oil-immersion objective (Figure IV). Their morphology can be seen even more clearly in a smear prepared from scrapings of the granuloma (Figure V). They are ovoid bodies 2 to 4μ in length, which have an eccentric circular nucleus and a paranuclear chromatin bar. They do not possess a capsule, which distinguishes them from *Histoplasma capsulatum*. The granuloma of histoplasmosis may bear a very close resemblance to that of cutaneous leishmaniasis in other respects. I have always found them as easily identifiable in hæmatoxylin and eosin stained sections as with Giemsa or Leishman stain. The latter type of stain is preferred for smears.

The main diagnostic difficulty is in the sections of an old lesion which has assumed a tuberculoid form, and in which the parasites are rare. The histological appearance is then indistinguishable from lupus vulgaris. Tuberculoid leprosy can usually be excluded because of the lack of involvement of nerves in the process, and tertiary syphilis by the absence of conspicuous endarteritis and plasma cells in the infiltrate. In all these conditions there is great difficulty in demonstrating the infecting organism. However, in patients who come from an endemic area, cutaneous leishmaniasis should be strongly suspected in any chronic granuloma, and must be excluded by a most careful search for the organisms before an alternative diagnosis is made.

[1] For Figures III-V see art-paper supplement.

Summary.

Five cases of cutaneous leishmaniasis in post-war Italian migrants to South Australia are reported.

The clinical and histopathological features of the condition are described.

The disease should be suspected in any migrant from a country where the disease is endemic who has a chronic granuloma of the skin.

Residence in Australia for several years does not exclude the possibility of the disease, because the incubation may be prolonged.

Acknowledgements.

I am indebted to the clinicians who allowed me to examine their patients and gave permission for the case reports to be published.

References.

DONALD, G. F. (1958), "Cutaneous Leishmaniasis (with Report of a Case)", *Aust. J. Derm.*, 4: 136.

LAST, J. M. (1960), "The Health of Immigrants: Some Observations from General Practice", MED. J. AUST., 1: 158.

Legends to Illustrations.

FIGURE III.—Dermal granuloma, extending from just below the epidermis to the subcutaneous fat. There are large numbers of histiocytes in the infiltrate, the paler areas being composed exclusively of such cells. The dark cells are mainly lymphocytes. The connective tissue has been replaced by the infiltrate (Case V). (Hæmatoxylin and eosin stain, x 148.)

FIGURE IV.—The superficial part of the granuloma showing macrophages containing numerous parasites (Case V). (Hæmatoxylin and eosin stain, x 1390.)

FIGURE V.—Smear of scrapings from an ulcer. Numerous parasites have been liberated from the cytoplasm of a ruptured macrophage (Case III). (Giemsa stain, x 1455.)

WHAT SHOULD THE CANCER PATIENT BE TOLD?[1]

By SIR WILLIAM UPJOHN, M.D., M.S., F.R.C.S., F.R.A.C.S., *Melbourne.*

THE question is often raised: "What should the doctor tell a cancer patient?" It is almost as important to consider how to tell the patient, as well as what to tell. We must remember that these people are fearful, that they have not the knowledge that medical practitioners have, that they may have misconceptions about the disease, which they have picked up in conversation or in reading magazine articles.

One might say a few words at this stage about the patient who comes to the doctor fearing she or he has cancer, but who is really quite free from it. It is very wrong to ridicule these people, to tell them that they "have cancer on the brain" or that they are wasting the doctor's time. Our anti-cancer campaign is directed to getting people to see a doctor if they have certain disquieting signs or symptoms, and if they meet with a discouraging rebuff when they do so for something which is unimportant, they will be ashamed and will be discouraged from again reporting when there is a real cause for them to seek medical advice.

In his undergraduate training the student learns much about the diagnosis and treatment of disease; but in

[1] Read at the Victorian Cancer Congress, Melbourne, August 22 to 26, 1960.

the institutional atmosphere of public hospitals he sees little example from his teachers of the need for regarding each patient as a separate personality requiring a humane and studied approach in order to give the best advice about his malady.

It is the duty of the physician or surgeon to consider each patient as an individual, to try to understand his personality, his way of life, his domestic and familial responsibilities, and to think upon these matters before deciding what it is necessary or desirable to tell the patient about his malady.

Generally, it will be best to tell the patient what is likely to be ultimately of benefit to him concerning diagnosis, treatment and his future prospects. In doing so, it is not proper to dwell on any gloomy features, but if anything hopeful can be said, to do so clearly and even repeatedly if this is justified.

It is not right to inform a patient that he has carcinoma if there is a possible doubt on the certainty of such a diagnosis. It is right to advise the patient to have tests, a consultation or even an operation, to establish beyond doubt that the malady is really carcinoma and not some condition closely mimicking carcinoma but requiring different treatment and having a different future course.

At the present time it is the practice to check the clinical diagnosis by taking a small piece of the supposed cancer and having this examined microscopically. Even this procedure is not free from error, because unwittingly a typical sample of the growth may not have been taken for microscopic examination.

Sometimes no useful object is attained by telling the patient the nature of the ulcer or tumour.

In old people malignant disease may be very slow in its progress. It may be controllable by means other than surgical operation, or the patient may have some other malady which will terminate life before the cancer can become troublesome. In this case no good and some mental discomfort may come through telling the whole truth.

It is not right to misstate deliberately the nature of the disease; but quite commonly a patient may not be anxious about the name of his disease — he is more interested in what can be done about it.

Of course, it may be necessary in some forms of cancer to tell the patient what he has got, because a surgeon cannot expect a patient to consent to undergo some major change in his anatomy, such as amputation of a limb, a radical major amputation of the breast, a removal of the rectum with the establishment of an "artificial anus" or colostomy and suchlike operations, unless he knows that he or she has cancer and that anything less than a radical extirpation of the affected part is worse than useless. It is a shocking thing for a patient to wake from an anæsthetic and find that extensive operative changes have been made in his anatomy, without having been warned before operation that this was contemplated by the surgeon. Explanation should be clear, but not gruesome, before the patient's assent is asked to what he regards as a mutilating operation. If he recoils from such a suggestion it is right to talk to him and remove any misconceptions he may have about being able to live a comparatively normal life after such surgery; but I do not think it is a surgeon's duty to urge a patient, particularly an elderly patient, to undergo some operation abhorrent to him in its effects. It should be enough for the surgeon to indicate clearly the benefits likely to follow operation and to correct any erroneous beliefs held by the patient. There is no need to go into unnecessary details; such explanations may be misunderstood or may be frightening or depressing.

On the whole, it will be found that patients are more likely to cooperate with the surgeon or radiotherapist if they have an adequate understanding of the serious nature of their disease and have not been misled by being allowed to think that their trouble is just an ulcer or cyst or warty swelling.

When for some reason or other a patient gets to know that he has a cancer past curing he will often be found to become more resigned to his troubles and will cease to be mentally agitated because he feels that he is not receiving treatment of a curative nature.

As much as possible, the medical attendant should avoid giving a definite prediction about the course and duration of the disease. Diagnosis and treatment have their difficulties; but prophecy even by the most experienced doctor may be wildly wrong. Those with an apparently hopeless prognosis may live on, and those with a favourable outlook may disappoint.

If the doctor is obliged by business or family circumstances to give a prediction to the patient, it is better to state what is known to be the average or usual prospect in his type of disease, and if the doctor knows of instances in which the survival and course of the disease have been much more favourable than the average, he should tell the patient, for it is remarkable what hope and faith in improvement may do for a patient. After all, it is an old and true saying, "While there's life there's hope".

THE LOW POSTERIOR APPROACH TO THE HIP JOINT: ITS APPLICATION TO PROSTHETIC ARTHROPLASTY.[1]

By ANTHONY HODGKINSON, F.R.C.S., F.R.C.S.E.,
Sydney.

THE "low posterior approach" to the hip joint is the more accurate description of the so-called "southern approach". This latter name, perhaps, has a greater "Confederate sympathy". Austin T. Moore, of Columbia, South Carolina, U.S.A., has popularized this approach to the hip with the use of his metallic replacement stem-type prosthesis. He developed its application in 1950; but Marcy and Fletcher previously had described its use for fracture-dislocations, and for access to tumours along the posterior aspect of the femoral neck.

This paper presents the results of the method as used by myself during the past three years in a limited series of 35 cases.

The Moore stem-type vitallium prosthesis was used because the Judet types are now considered mechanically unsound. The series covers age groups ranging from 56 to 96 years; the conditions involved included osteoarthritis, fresh subcapital fractures, old subcapital fractures, "failed" Judet acrylic prostheses and "failed" Smith-Petersen pins. Austin Moore's operative method is slightly modified to give better sciatic nerve protection.

Pre-Operative Preparation.

Blood Requirements.

One litre of blood is ordered for patients with fresh fractures, and 1·5 litres for osteoarthritic patients, as standard practice. It is not often that more than 500 ml. is required for fresh fractures.

Skin Preparation.

The skin preparation is important because of the incision's proximity to the anus. The mechanical inaccessibility of the region when the patient lies in bed with a hip condition makes operation-room skin preparation the only satisfactory method.

A thorough eight-minute rub-down of the operative field from the knee to the iliac crest level is conducted with sterile gloves. For obvious reasons, this can be done more briskly when the patient is anæsthetized. At present a hexachlorophene preparation is used. The skin is dried, and fresh iodine in 80% alcohol is painted gently over the

[1] Read at a meeting of the Australian Orthopædic Association on October 12, 1959, at Melbourne.

region. A warning is given regarding iodine sensitivity, as shown by Figure I. It shows what happened when iodine was used to rub a swollen knee. Abdominal skin testing with iodine 48 hours before operation is a routine precaution which is worth taking.

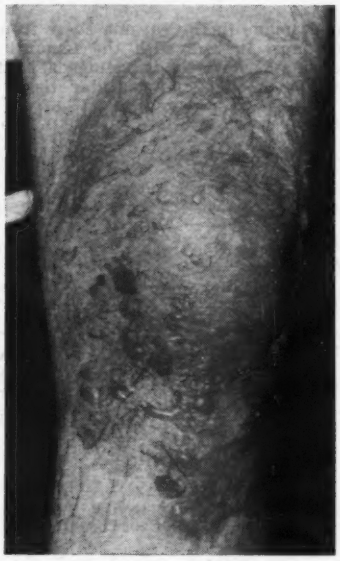

FIGURE I.
Photograph of the skin of the knee after it had been rubbed with iodine.

Method of Sterile Draping.

The patient is draped in the semi-prone position, as shown in Figure II. Support can be maintained with sandbags or a pubo-sacral clamp, or just wide adhesive strapping. Care is needed not to restrict respiratory excursion with the

FIGURE II.
Showing the posture.

latter method. The leg concerned must be free to be adducted to the level of the table, and free to be flexed to 70°, as shown in Figure III. The draping must be complete from the foot to above the knee, and around the natal cleft to the pubis. The iliac crest is exposed and the leg concerned is free. An abdominal sheet with an 18 inch central split forms a firm fit around the perineum and thigh.

Incision and Surgical Anatomy.

The incision begins at a point four fingers' breadth below and posterior to the posterior superior iliac spine, as shown in Figure II. It passes in a curved line distally and laterally to lie two fingers' breadth posterior to the crest of the greater trochanter of the femur. The scar thus avoids pressure from the prominent trochanter crest. The deep fascia opens on to the gluteus maximus muscle at the junction of the middle and distal thirds of its mass. Figure IV again shows its relative position.

The incision level can be varied in its distance from the posterior superior iliac spine. It is an advantage to begin less than four fingers' breadth inferiorly if there is proximal shortening of pericapsular tissues. This occurs in old subcapital fractures to which traction has not been

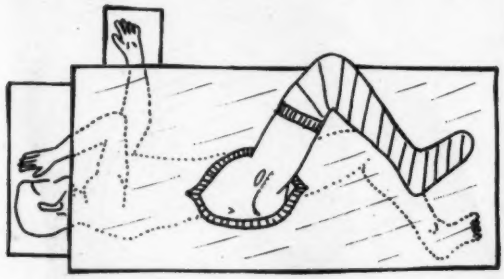

FIGURE III.
Drawing of draping.

applied, or when pins have been unsuccessfully inserted, or in osteoarthritis. Figure V demonstrates an osteoarthritic type incision.

The incision can also be extended distally below the greater trochanter if increased exposure is required. Here the incision passes through the gluteus maximus aponeurosis in line with its superficial fibres which are inserted into the fascia lata. Therefore the extension does not cut across deep or superficial fibres.

FIGURE IV.
Drawing of posture from behind.

Exposure of the Sciatic Nerve.

At this point, side towels are sutured or clipped into position as a further protection against excessive skin handling. Blood ooze is controlled with diathermy coagulation. The immediate deep relation of the sciatic nerve to the gluteus maximus is kept in mind, and the display of the nerve is begun from the distal gluteus maximus aponeurotic tissues. On opening into the areolar layer beneath the muscle, the coarse fibres are split gently towards the proximal end of the skin incision. This is done by digital separation of the fibres. The sciatic nerve with its artery and vein is usually readily visible through a thin areolar layer. Variations occur when there is high division into medial and lateral popliteal nerves. Also, there is often an extensive varicosity on the nerve, resulting from an enlarged sciatic vein. Figure VI shows

an actual operative exposure. The sciatic nerve is shown crossing the lower part of the wound, lying on the short external rotator muscles. Anatomically, most of the large vessels and the nerve supply enter the gluteus maximus from the supero-medial aspect of the muscle. The nerve divides into roughly two main divisions before its muscle ramifications. These are spared with this technique. If

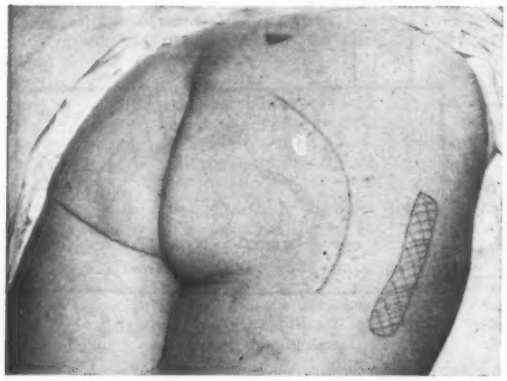

FIGURE V.
The incision.

the muscle fibre-splitting is not carried too far superiorly and medially, no excessive bleeding occurs.

The areolar fat is gently eased aside, any excessive handling of the sciatic nerve being avoided. Neuropraxia can result from manipulation of the nerve, and bleeding from the nerve vessels can be a trouble.

Posterior Exposure of the Hip Joint Capsule.

The short external rotators of the femur are well displayed when the areolar tissue is removed from their

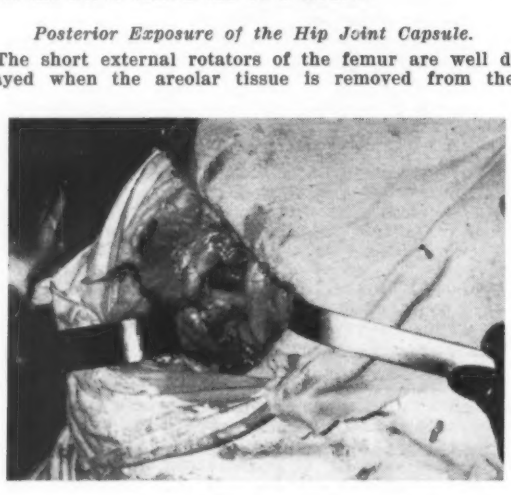

FIGURE VI.
Sciatic nerve exposure.

surface. Figure VII diagrammatically shows the relationships. The short external rotator muscles vary slightly in their presentation. This is because attachments to the femur vary anatomically, and proximal joint tissue contraction often occurs with hip disease.

The next step is to incise these muscles at their femoral attachment, and secure the ends with long chromicized catgut ties. They strip away from the posterior surface of the capsule, but there is slightly more oozing of blood when osteoarthritis is present. A constant bleeder occurs in the substance of the quadratus femoris muscle, this being the ascending articular branch from the medial circumflex artery (Figure VIII). Traction on these muscles over the sciatic nerve protects the nerve, as this drawing demonstrates. It establishes a definite plan by which one can watch for the nerve without excessive traction during the more forceful manipulations of the operation.

An incision is made in the capsule away from the nerve towards the femur, in line with the femoral neck. The acetabular labrum posteriorly is identified, and a circumferential incision through the capsular tissue is carried

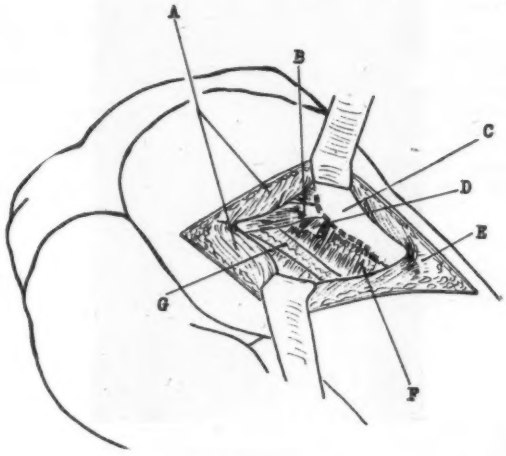

FIGURE VII.
Superficial wound: A, split gluteus maximus and bleeding vessels; B, pyriformis; C, greater trochanter; D, obturator internus with gemelli; E, aponeurosis of gluteus maximus; F, quadratus femoris and bleeding vessels; G, sciatic nerve and vein.

around to the anterior aspect as far as possible. Osteoarthritic thickening, calcification and oozing of blood make this stage more difficult than with simple fractures. However, it can be done adequately. As much as possible of the capsular tissue is excised, and any bleeding is controlled with coagulation diathermy. This helps to suppress post-operative osteophyte formation.

Posterior Dislocation and Removal of Femoral Head.

The hip disease is adequately exposed at this stage. Removal of the mobile head in the subcapital fractures, and mobilization of the intact head and neck prior to posterior dislocation, are facilitated by the use of the Smith-Petersen crank-handled curved gouge (Figure IX). It is introduced into the acetabulum to cut the ligamentum teres, and with traction it is possible to extract a loose femoral head. Also, it assists in dislocating posteriorly a diseased intact head and neck. This latter manœuvre is important, because it relieves the tendency to use too much torsional force from the internal rotation on the femoral shaft through the knee joint. This torsion can produce a supracondylar femoral shaft fracture if the bones are osteoporotic, or can cause damage to the ligaments of the knee.

Display of the acetabulum and the femoral neck is now possible by adduction, internal rotation and proximal dislocation of the femoral trochanter. Figure X shows the extent of the internal rotation which is possible once the short external rotators and capsular joint tissues have been freed and the femoral head has been dislocated posteriorly from the acetabulum.

Further adduction of the thigh with the knee to the level of the operating table, and then proximal dislocation of the greater trochanter over the superior portion of

the gluteus maximus muscle mass, present the femoral neck and acetabulum as shown in Figure XI. The femoral head can now be easily excised from the neck if required. Adequate relations can be assessed for remaining neck length by visualizing the lesser trochanter. Further

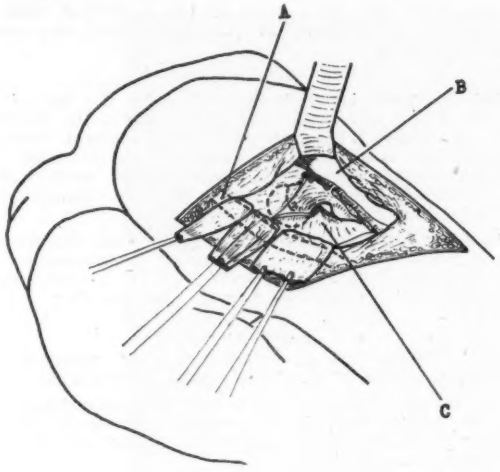

FIGURE VIII.
Exposure of the posterior hip capsule: A, sciatic nerve; B, greater trochanter; C, incised capsule.

extensions of the capsular excision can also be carried out. The acetabulum can be inspected for any irregularities, for complete excision of the ligamentum teres, and for control of any bleeding from its artery if it is patent. Before the femoral neck is trimmed for the prosthesis, a gauze strip is packed into the acetabulum to prevent bone chips from collecting in the blood clot. The neck is trimmed to the right length, and the prosthetic rasp is introduced as shown in Figure XII. Adequate room is available in this method for avoiding the soft tissues during reaming, and to provide the correct angle of anteversion for the prosthesis in the femoral neck.

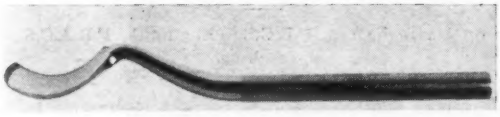

FIGURE IX.
Smith-Petersen cranked curved gouge.

Insertion of Prosthesis.

The prosthesis head size is measured from the original femoral head if this is available, and a trial is made before its insertion. The prosthesis is fitted without force into the femur, and the bone chips are inserted into the fenestrations before it is finally pushed home. These chips are perhaps more necessary in the long-term cases.

After satisfactory siting of the prosthesis stem, the acetabulum is dried, the sciatic nerve is again identified and reduction of the head dislocation is carried out. The knee is extended; gentle antero-lateral digital pressure on the prosthetic head and coordinated extension and traction of the thigh enable the reduction to be easily and safely carried out (Figure XIII).

The short external rotator muscles are then returned loosely to their femoral attachment. This is facilitated by externally rotating the leg. They offer no strength or muscle stability, but help to form a living cushion of muscle between the metal prosthesis and the sciatic nerve. This feature is well demonstrated by the tension of the nerve across these muscles when the prosthesis is fitted into a limb in which capsular shrinkage has taken place in an old fracture. The fibres of the gluteus maximus fall together and may be approximated by a few loose catgut sutures. The muscle belly thus forms a

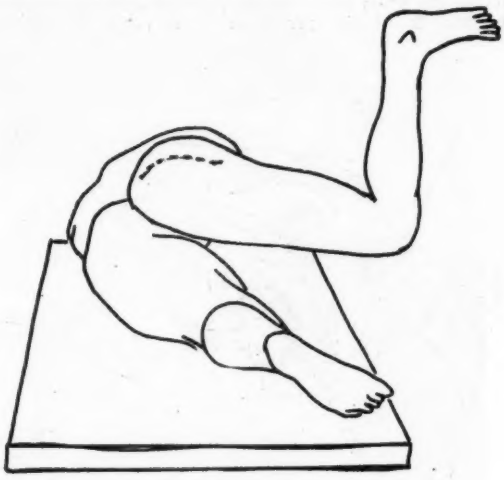

FIGURE X.
Internal rotation after capsulectomy.

healthy vascular barrier between the hip and the skin. This aids the prevention of infection complications.

Discussion.

The mechanics of this exposure allow the patient to be free in bed and to move the leg without any fears

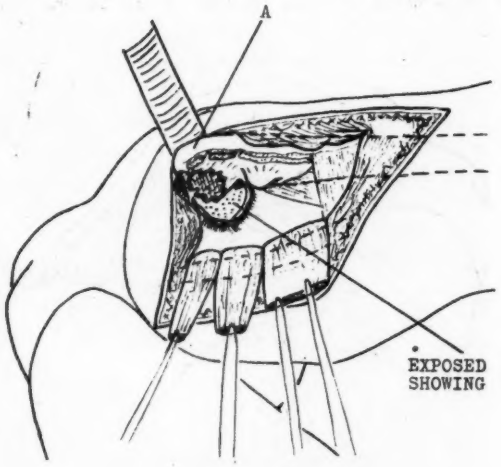

FIGURE XI.
Femoral neck dislocation in wound: A, greater trochanter dislocated proximally out of wound and neck rotated out by internal rotation; B, exposed acetabulum, showing excised capsule.

of dislocation. For the elderly, it is a benefit to their well-being to get up, sit out and even to stand on the leg. This can be done the next day with a primary fracture prosthesis. It is a feature which proves extremely helpful with a hemiplegic, or if the patient is blind. These

examples came from two patients on whom I operated recently, one being aged 75 years and the other 92 years.

There are certain dangers in this approach. The sciatic nerve can cause anxiety with the exposure. I have seen it reduced into the acetabulum with the prosthesis. The anteversion must be allowed for, before one can expect a stable hip when such radical capsular excision is carried out. The fit of the prosthetic stem must be easy but

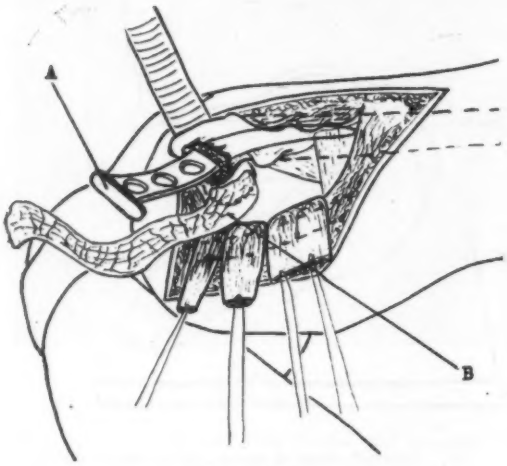

FIGURE XII.
Moore rasp in position: A, rasp; B, gauze roll packed into acetabulum.

firm, without toggle. No force must be used to tap it home, or the femur will split just as a wedge splits a piece of wood. No force must be used with the dislocation by internal rotation or with reinsertion of the prosthetic head into the acetabulum. Adequate muscle relaxation at this stage helps to avoid fracture of the femoral shaft

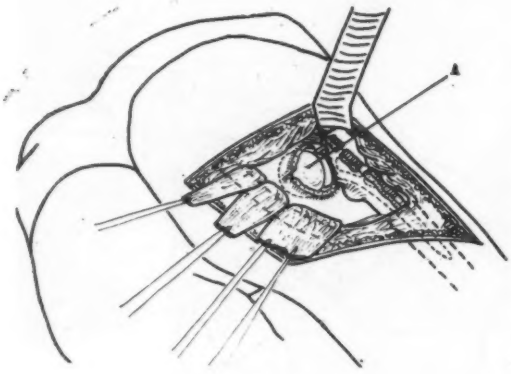

FIGURE XIII.
Prosthesis in position (A).

and neck. Internal rotation causes a twisting of the soft tissues of the leg. This can produce venous congestion. A femoral clot could be dislodged if one is present, as may be the case if there is delay in operation on elderly fracture patients.

Despite these disadvantages, this exposure has been most successful in this limited series. There has been no infection and no post-operative dislocation. Three deaths have occurred. One was that of a man, aged 80 years, who succumbed 10 hours after operation from pulmonary embolism. His operation was delayed seven days from the time of his fracture. Another was that of a woman, aged 70 years, who died 48 hours after operation from a coronary occlusion. She had had an ununited subcapital fracture in her left hip for 24 weeks, and had remained in bed for ten weeks after sustaining a subcapital fracture in her right hip. The third patient, a woman, aged 93 years, died from congestive cardiac failure two weeks after operation.

Conclusion.

This exposure is admirably suited for the use of the Austin Moore prosthesis. The approaches to the hip joint by the anterior, lateral and postero-lateral methods necessarily sacrifice hip-stabilizing muscle and the ligamentous tissues. Such exposures must mean a more prolonged operative procedure and less efficient post-operative convalescence.

The low posterior approach preserves the integrity of the essential hip-stabilizing anatomical structures, and allows a complete operation of moderate severity to be done on a wide range of patients with less risk.

Summary.

A description of the "low posterior approach" or "southern approach" to the hip joint is presented.

The details of the operation and its application in the use of the vitallium Moore stem-type prosthesis, as was originally described by Austin T. Moore of Columbia, U.S.A., are outlined.

The results of its use in a series of 35 cases describe its advantages, versatility and possible complications.

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PRE-OPERATIVE AND POST-OPERATIVE INTRAVENOUS THERAPY: THE RECOGNITION AND TREATMENT OF A DISTURBED FLUID AND ELECTROLYTE BALANCE.[1]

By DAVID FAILES, F.R.C.S. (England), F.R.A.C.S.,
Sydney.

A PATIENT may be out of water and electrolyte balance when he is first seen, or his imbalance may develop during the course of treatment. There may be an excess or a deficiency of water or electrolytes in the body. How is such a condition managed?

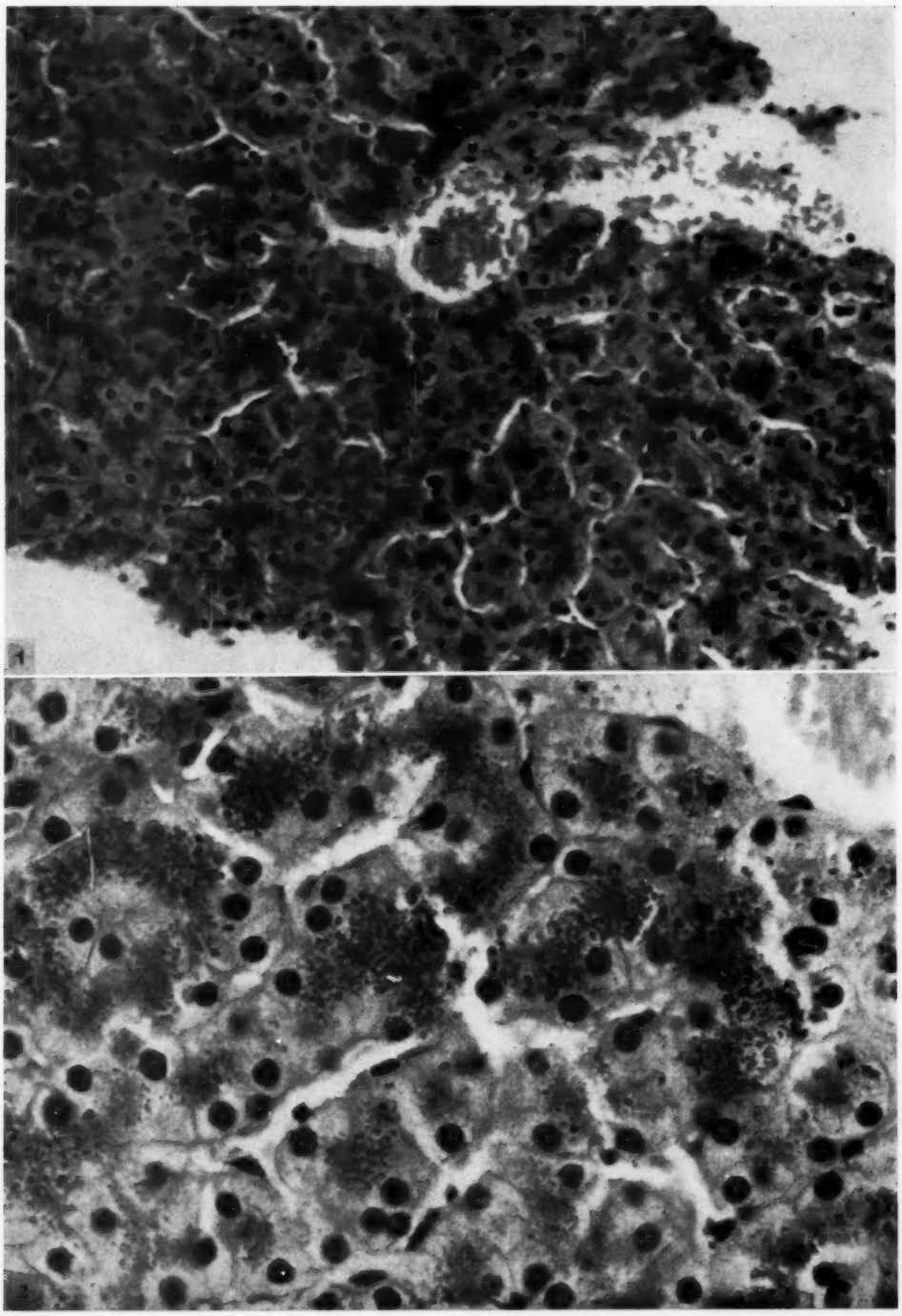
The first step is recognition and assessment of the imbalance; this depends on the history, physical examination and biochemical estimations.

It is of the greatest importance to obtain a history of any excessive or inadequate intake or loss, and to determine the period during which this has been occurring, in order that some quantitative estimate of such gains and losses may be made. Further, it is important to know what type of fluid is being gained or lost — for example, vomitus, or loss by fistula or by diarrhœa. Any available fluid-balance data should be studied. The patient's complaints as to thirst, weakness, faintness or fatigue should be ascertained. Inquiry should be made into the patient's past health.

A thorough physical examination should be carried out, with particular reference to the general state of the patient, physical and mental, the patient's weight, the pulse rate, the blood pressure and the state of the

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 28, 1960.

ILLUSTRATIONS TO THE ARTICLE BY J. B. COPE AND A. W. STEINBECK.



ILLUSTRATIONS TO THE ARTICLE BY K. V. SANDERSON.

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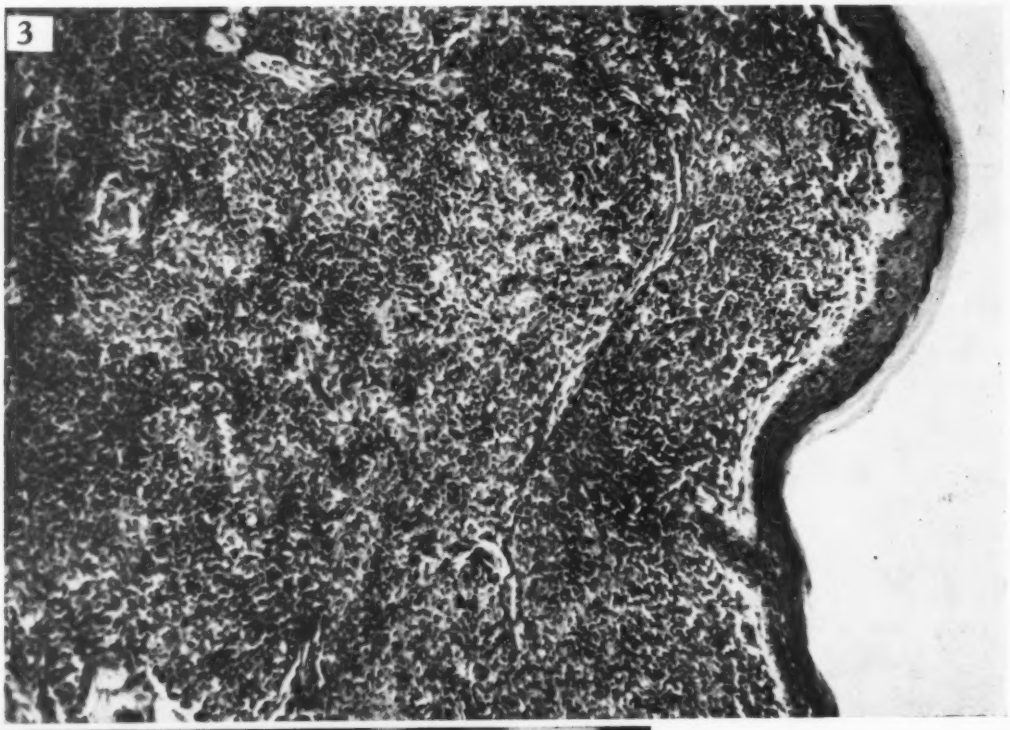

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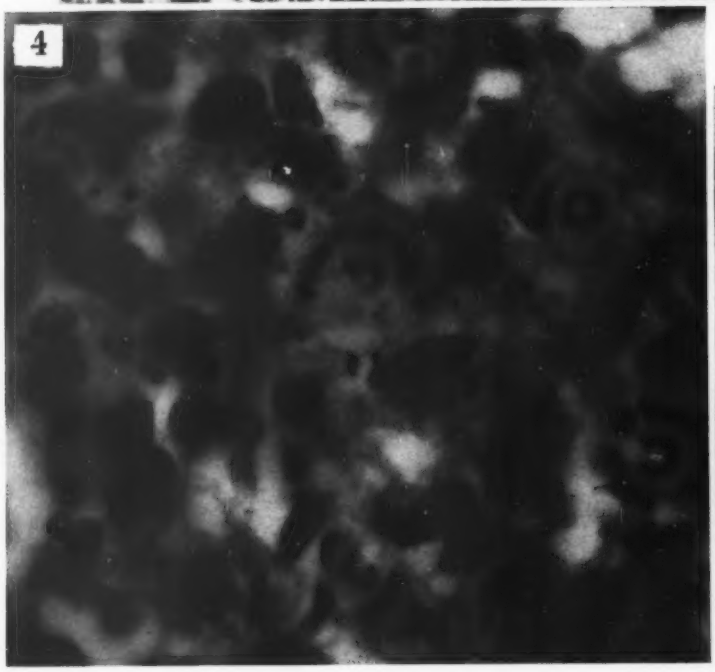

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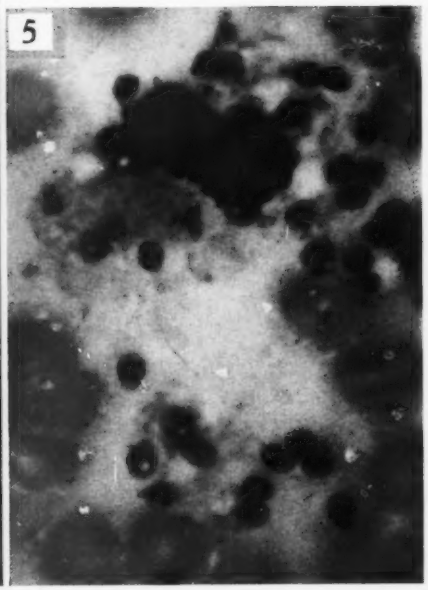

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veins, dryness of the mouth, tension of the eyeballs and elasticity of the tissues.

The volume of any urine passed, together with its specific gravity, should be recorded, in addition to the usual tests for sugar and albumin.

Laboratory tests are essential. Estimation of the hæmoglobin value or hæmatocrit and of the serum electrolytes should be performed at the outset to give a baseline for treatment; these tests should be repeated at intervals, usually daily, to note progressive changes. Such other tests as estimation of the electrolyte concentration of urine and aspirated fluid, the plasma protein content and the blood urea and creatinine content, together with an electrocardiogram, may be necessary.

From the foregoing clinical and biochemical findings, an estimate of the type and quantity of fluid and electrolytes lost or gained should be made, and the effect on the body of such gains or losses should be assessed. In addition, it should now be possible to postulate whether the body's "controlling mechanisms" are normal. If there is any evidence of abnormal control mechanisms, such as impaired renal function, then the problem is a most difficult one, requiring individual care and expert knowledge; such a patient ideally should be transferred to a specialized unit for treatment.

It should be borne in mind that intravenous therapy is chiefly an adjunct to surgical and medical treatment; the doctor must not become so preoccupied with this therapy as to overlook the need for operation or other definitive forms of treatment.

With regard to management, some general principles may be laid down.

Really accurate fluid record charts are essential; the ideal method is to collect all fluids lost from the body in large jars, so that every 24 hours the volume of each may be measured and the electrolyte concentration determined.

As treatment proceeds, repeated clinical examination is necessary; very ill patients require reassessment at very frequent intervals, and a severely shocked patient requires constant supervision. Intravenous therapy carries certain risks, such as the danger of over-transfusion, and the main safeguard against these risks is close supervision of the patient.

The treatment of a deficiency is to supply the missing substances, the quantity being judged from an estimate of the losses. Provided that the kidneys are normal, it is better to give more rather than less and to allow the kidneys to make the necessary adjustments.

The treatment of excesses is by one of three methods: (*a*) to restrict further intake so that the balance may be regained over the next few days; (*b*) to shift the excess from a dangerous to a less harmful place — for example, the temporary transfer of potassium out of the plasma into the cells by giving glucose with insulin; or (*c*) to remove the excess, either slowly (for example, removal with ion-exchange resin) or more rapidly by dialysis on the "artificial kidney".

In surgical practice, a disturbed balance is nearly always due to loss of body fluids, and thus the common problem is one of a deficiency of fluid and electrolytes. However, less commonly, when abnormal renal function complicates intravenous therapy, the problem may be one of an excess of water and electrolytes. Thus the following clinical states may be considered — those due to loss of body fluids and those due to abnormal renal function.

Clinical states due to loss of body fluids may result from hæmorrhage, or from some abnormality in the alimentary tract, as in intestinal obstruction, pyloric stenosis, diarrhœa or fistulæ (external). They may also be due to sequestration of fluid, as in burns and peritonitis.

Clinical states due to abnormal renal function may result from acute renal failure, chronic renal failure or uretero-colic anastomosis. Disturbances also occur in various other "medical" conditions — for example, nephrotic syndrome, congestive cardiac failure, steatorrhœa, cirrhosis, Cushing's syndrome, Addison's disease and after steroid therapy.

Losses Due to Hæmorrhage.

Acute blood loss results in oligæmic shock, and the treatment is to give whole blood. If blood is not available, then serum albumin, or even "Dextran", may be used. The subject of blood loss and its treatment has been discussed by the previous speaker.

Intestinal Obstruction.

It is proposed first to consider intestinal obstruction without strangulation.

What is the composition of the fluid which is lost in intestinal obstruction? The electrolytes lost will vary according to the level of the obstruction. In infants and young children, the composition of the intestinal fluids may vary greatly; however, in adults the fluid lost may be considered to be approximately equivalent to plasma with slightly increased potassium content. There is loss of sodium and chloride ions, of which sodium is in excess. The continued loss of sodium will lead to severe depletion, with reduction of the extracellular volume and circulatory collapse. Thus the replacement fluid should be normal saline, with potassium added as necessary. If the patient is clinically "shocked", then in addition, serum albumin should be given until the blood pressure has been restored.

How much saline should be given? An estimate of the quantity of the deficit should be made; however, the actual amount given must be judged by the clinical response to the intravenous therapy. There is no formula or plan which can be used as an absolute guide. The intravenous administration of fluids should be continued until the circulation has been restored, as judged by the return of the pulse and blood pressure to normal levels, the return of warmth to the extremities and the presence of normal or slightly increased filling of the neck veins. The amount required in an adult of average build with signs of severe dehydration — rapid pulse rate, low blood pressure, dry inelastic skin, scanty urine of high specific gravity — is likely to be of the order of four to five litres.

When a sodium, rather than a water, deficiency exists, then hypertonic (3%) saline may be used.

In addition to sodium and chloride, it may be necessary to give potassium intravenously. The clinical signs of potassium deficiency are lassitude, weakness and mental apathy. Delay in the return of bowel sounds after gastro-intestinal surgery may be a sign of potassium deficiency. However, the diagnosis really depends on the estimation of the serum level of potassium. With a moderate potassium deficiency — that is, a serum potassium level above 3·0 mEq/l., it is advisable to restore the circulation and to reestablish the urinary output before giving potassium. When intravenous potassium administration is commenced, certain precautions should be observed; there must be an adequate urinary output (at least 500 ml. per day); it should be given in a suitable concentration (maximum concentration 80mEq/l.); it should be given at an even rate, but not at an excessive rate (maximum rate 20 mEq/hour) and it is desirable that the serum potassium level be estimated daily.

Potassium may be given either by adding ampoules containing 0·75 gramme of potassium chloride in 10 ml. to a 5% glucose solution (each millilitre of the potassium ampoule contains 1 mEq. of potassium), or by the use of prepared solutions, such as Darrow's solution (which contains 35 mEq/l of potassium) or a 0·6% solution of potassium chloride in 5% glucose solution (which contains 80 mEq/l. of potassium). The administration of glucose with the potassium is of value, since it tends to shift the potassium into the cell, and helps

to prevent the occurrence of high serum levels should the potassium solution be given too rapidly.

However, when losses have been excessive and prolonged, there may be a severe potassium deficit at the outset. In such a case, the serum potassium level may be as low as 2·0 mEq/l.; such levels suggest that the total body deficit of potassium is of the order of 300 to 500 mEq. Such a condition is extremely dangerous, and potassium administration should be commenced immediately the severe deficit is revealed.

Once the deficit of fluid and electrolytes has been made good, further intravenous administration is usually necessary to replace continuing "abnormal losses" from the alimentary tract, and also to replace "normal losses" in the urine and fæces and from skin and lungs. In an adult, the "normal losses" may be replaced daily by 2·5 litres of 5% glucose solution in fifth-normal saline.

When intravenous therapy has to be maintained for several days, it is important to provide adequate calories to minimize the breakdown of body protein. If the patient can take plenty of fluid, a 10% glucose solution may be used; this yields 400 Calories per litre, and thus 3 litres per day would supply 1200 Calories. When the volume of fluid must be restricted, more concentrated solutions are required (up to 50% glucose); since such solutions cause severe thrombophlebitis in superficial veins, they must be given via a "Polythene" catheter into the inferior vena cava. A recent innovation in this field has been the development of solutions of fat emulsions for intravenous therapy; a 15% emulsion of olive oil has been used, providing 1200 Calories per litre. Mild reactions, such as chill, back pain, fever, nausea and vomiting, chest pain and generalized cramp, occurred in one-quarter of patients receiving such transfusions; thus it is recommended that the use of these solutions be limited to 1 litre per day; indeed, some surgeons are opposed to their use.

The presence of strangulation will modify treatment in two ways. First, blood may be necessary as the replacement fluid, since large amounts of blood may become sequestrated into the strangulated bowel; secondly, resuscitation must be carried out more rapidly in view of the urgency of operative treatment; in a severe case, it is suggested that a maximum of two hours be allowed for resuscitation prior to operation.

Pyloric Stenosis.

In pyloric stenosis, the loss of gastric juice entails considerable loss of hydrochloric acid and gastric mucus. The chief loss is the chloride ion. However, the gastric mucus contains sodium and a small amount of potassium; thus there is an amount of sodium lost to the extent of about half the chloride loss. The loss of chloride leads to a rise in the sodium bicarbonate level, so that a metabolic alkalosis develops. As a consequence partly of this alkalosis and partly as a result of external losses, the serum potassium level falls.

Thus the effect of pyloric stenosis on the body is to cause a lowering of the serum levels of sodium and chloride, especially the latter, a considerable rise in the serum bicarbonate level and a fall in the serum potassium level.

The condition is treated by giving ample amounts of normal saline (5 litres or more if necessary) and allowing the kidneys to make adjustments, discarding the unwanted excess. As the deficits in sodium and chloride are corrected, the serum bicarbonate level will fall, and this leads to a rise in the serum potassium level. In some cases. additional potassium may be required. As a general rule, it is not necessary to resort to the use of ammonium chloride.

External Fistulæ.

In external fistulæ involving the small bowel, the effects of loss of fluid via a fistula are, in general, similar to loss of fluid by vomiting or aspiration as in intestinal obstruction. However, at times the fluid lost by a fistula may be more selective; for example, in a pure pancreatic fistula, the fluid lost will contain a large amount of bicarbonate (60 to 80 mEq/l.) in addition to the usual large amount of sodium.

Management is along similar lines to intestinal obstruction. The fluid lost should be collected by inserting a catheter into the fistulous track and applying suction drainage to the catheter; this collection enables the volume and electrolyte composition of the fluid to be measured. The fluid is replaced by normal saline, and if necessary by potassium as well. When a pancreatic fistula is present, the serum bicarbonate level must be carefully observed, and any tendency to acidosis is checked by giving sodium lactate.

Diarrhœa.

In acute diarrhœa, the fluid lost resembles that which passes from the small intestine into the large, containing considerable amounts of sodium and chloride and some potassium. The effects of this loss and its treatment are similar to those in intestinal obstruction.

In chronic diarrhœa, as in chronic ulcerative colitis or the overuse of laxatives, there is persistent loss of fluids that are disproportionately high in potassium content. The end result of this is a severe potassium deficit with a lower serum potassium level. Treatment is to give large amounts of potassium salts and also some normal saline.

Peritonitis.

An example of fluid loss due to sequestration of fluid is in peritonitis. In this condition, there will be accumulation of fluid, electrolytes and protein in the peritoneal exudate; as in burns, protein is lost from the plasma, leading to hæmoconcentration and circulatory collapse. The condition is aggravated by losses from the alimentary tract, which will also be occurring when peritonitis is present.

Management follows the general principles already discussed, with emphasis on the addition of serum albumin to the replacement fluid and the careful observation of the hæmatocrit or hæmoglobin readings to check any tendency to hæmoconcentration.

Acute Renal Failure.

Up to now, consideration has been given to the disturbed balance resulting from abnormal losses of fluid and electrolytes from the body. However, the opposite problem may arise as in acute renal failure, in which abnormal renal function results in an excess of fluid and electrolytes in the body.

Acute renal failure means that the urine output is insufficient to enable the kidneys to excrete all the waste substances; thus substances which should have been excreted are retained in the body. This implies that the urine output is less than 400 ml. per day; the characteristic urine volume in acute renal failure is 150 to 300 ml. per day. The common causes of this condition are circulatory insufficiency and hypotension, intravascular hæmolysis, renal disease and obstruction of the urinary tracts. The effect of renal failure is to cause severe uræmia and acidosis, high serum potassium levels and retention of sodium with over-hydration.

With regard to management, the first principle is prevention. Most of the causes are preventible — for example, early and effective treatment of shock and dehydration should prevent circulatory collapse and prolonged hypotension.

The next principle is early recognition. Any patient whose daily urine output after operation or trauma is less than 400 ml. should be suspected to be suffering from renal damage; if the oliguria persists despite an adequate circulation, then the diagnosis of acute renal failure should be made.

The aim of treatment is to keep the patient alive until spontaneous recovery of renal function occurs. The probable cause should be treated; intake should strictly

be controlled — 400 ml. of water per day are allowed — and food should be free from protein, sodium and potassium; the level of fluid and electrolytes should be carefully corrected. Dialysis on the artificial kidney may be necessary. Ideally, all patients suffering from acute renal failure should be transferred to a specialized unit, where expert knowledge and specialized equipment, such as an artificial kidney, are available.

With intelligent treatment, the majority of patients survive and the recovery of renal function is usually good.

Conclusion.

In conclusion, although there are still many gaps in our understanding of this difficult subject, I believe that the best approach to this problem is to attempt to assess the underlying pathological and physiological mechanisms in the individual case. Such knowledge then affords a logical basis for clinical management.

Acknowledgements.

I wish to thank Dr. H. M. Whyte, Director of Medical Research, Kanematsu Memorial Institute, Sydney Hospital, for his valued advice and assistance, and Dr. W. E. L. Davies, Resident Pathologist, Sydney Hospital, for most helpful discussions.

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METHYL CHLORIDE INTOXICATION.

By I. J. MACKIE, M.B., B.S., M.R.A.C.P.,[1]
Sydney.

METHYL CHLORIDE at temperatures above −23° C. is a colourless, transparent gas, quite non-irritating to the respiratory tract and having only a very faint, sweet odour.

Most of the reported cases of poisoning have involved people associated with commercial or domestic refrigeration plants; but the gas is also used in synthetic rubber and petroleum-refining industries as an extractant and as an agent for methylation and chlorination. Entrance to the body is principally by inhalation, but skin absorption is said to be significant enough to make the gas mask alone of little value in preventing intoxication.

Review of the Literature.

Eulenberg in 1896 first described the toxicity of methyl chloride in pigeons, but it was not until 1914 that humans were recorded as having been affected. Since this time, over 200 cases have been reported with some 20 deaths, and in all except one the clinical picture has followed a definite pattern with but minor individual variations. Vertigo, subjective weakness, blurring of vision, muscle incoordination, drowsiness and gastro-intestinal symptoms (especially nausea and vomiting) are almost invariable, while mental confusion, insomnia, nightmares, paræsthesiæ, hiccups, muscle tremors, ptosis and dysphagia occur very commonly. In the severe cases, opisthotonic spasms may occur, or else generalized epileptiform seizures alternating with periods of deep unconsciousness. Recovery in non-fatal cases is usually complete in 10 to 20 days, although sequelæ including foot-drop, ataxia, ptosis, blurred vision, diplopia and amnesia have all been noted and have lasted for up to a year after the acute episode. Autopsy studies have shown hyperæmia of lungs, liver, kidney and brain with subpleural and subepicardial hæmorrhages. Early degenerative changes have been noted in the liver, while numerous lipoid-filled histiocytes have been seen in the leptomeninges covering the hemispheres. The clinical and pathological features in the experimental animals of Smith, Dunn and von Oettingen bore a very close resemblance to those seen in humans. In 1951, Sperling, Macri and von Oettingen administered methyl chloride to dogs by intravenous injection, and studied its rapid disappearance from the blood-stream; pulmonary excretion in the first hour accounted for only 5% of the amount injected and the bile for less than 5%.

It is said to enter the cells of the body where hydrolysis to methyl alcohol and hydrochloric acid occurs; Flurry believes that methyl chloride is hardly toxic in itself, and that its effects are due to methyl alcohol and its oxidation product, formaldehyde.

Wood, in 1951, described a case quite different from the typical one, in a refrigeration engineer who admitted to having taken very few precautions to avoid inhalation of the fumes. At autopsy, this patient had a grossly cirrhotic liver some 18 months after an attack of mild jaundice lasting a few weeks. The author concedes that his case may, in fact, be one of cirrhosis following infectious hepatitis, but has good evidence for concluding that such is not the case.

Report of a Case.

The patient, an Australian from a country town in New South Wales, aged 40 years, was the owner of a small refrigeration plant, and on September 24, 1957, he was admitted to the Royal Prince Alfred Hospital, gravely ill. He had been perfectly well until about 12 months prior to his admission, when he suffered the gradual onset of sexual impotence, which became complete over a period of three or four months; nevertheless he felt in good health until May, 1957, after which he was never well. The long hours of strenuous work to which he was subjected became an increasingly great burden, and he felt weak, listless and somewhat apathetic and lost weight. Furthermore, his wife and friends commented on how pale he looked. Just six weeks prior to his admission to hospital, he had had an episode of "the 'flu", characterized by anorexia, nausea and vomiting, generalized weakness, muscle pains and elevation of temperature up to 102° F.; two days later clinical jaundice appeared and the systemic symptoms ameliorated. The urine became dark and the fæces soft and very pale. Over the succeeding six weeks, the jaundice deepened progressively, the urine remained dark and the fæces pale, anorexia persisted, and he commented on blurring of vision. After five weeks pruritus developed and lasted only four or five days, but during this time he had also a transient generalized erythematous rash which blanched on pressure. During this six-week period he developed, in addition, an absolute revulsion for both smoking and drinking, whereas he had formerly partaken enthusiastically of both. (He had been in the habit of taking five or six 10 oz. glasses of beer each day for many years, and smoked 20 cigarettes per day.)

On his admission to Royal Prince Alfred Hospital, in addition to the symptoms already enumerated, he complained of a throbbing bitemporal headache, thirst and polyuria, all of three days' duration, and also of a slight morning urethral discharge with some scalding and frequency of micturition of two days' duration. At this stage, he volunteered the information that for some months his urine had had the same musty odour that he associated with mice.

Physical examination of the patient revealed a normal temperature, a pulse rate of 120 per minute, deep jaundice and profound pallor of the mucous membranes. The liver edge was palpable two fingers' breadth below the right costal margin; it was soft and very tender, the spleen

[1] Formerly Medical Registrar, Royal Prince Alfred Hospital, Sydney.

TABLE I.

Date.	Serum Bilirubin Content. (Mg. per 100 Ml.)	Serum Alkaline Phosphatase Content. (K.-A. Units.)	Thymol Turbidity.	Zinc Sulphate Flocculation. (Normal, 2 to 6 Units.)	Serum Protein. (Grammes per 100 Ml.)	Blood Urea Content. (Mg. per 100 Ml.)	Result of Schumm's Test.
1957:							
September 24 ..	12·0	25	Absent.	4·5	6·3	54	Positive.
September 28 ..	9·7	26	+	4·5	—	—	Positive.
October 4	6·0	22	++	5·0	—	—	Negative.
October 14	6·0	31	++++	15·5	—	—	Negative.
October 20	3·8	25	++++	18·5	7·6	32	Negative.
October 25	—	—	—	—	8·0	—	—
October 29	3·4	22	+++	14·0	—	—	—
November 4	0·8	20	+++	17·0	7·6	—	—
1958:							
February 24	0·2	8	+++	7·5	—	—	—

was enlarged and palpable two fingers' breadth below the left costal margin. There were no other abnormal physical findings. His weight was 133 lb. The fæces were normal, but the urine contained bile pigments, bile salts and a heavy cloud of protein.

Over the next 36 hours, his temperature "spiked" between 97° and 104° F., and the following laboratory data became available: hæmoglobin value, 3·2 grammes per 100 ml.; white cell count, 47,000 per c.mm. (3% neutrophil myelocytes, 10% mature neutrophils and 70% segmented neutrophils); platelets, 1,400,000 per c.mm.; result of Schumm's test, positive; result of direct Coombs test, negative; blood urea content, 54 mg. per 100 ml.; serum protein content, 6·3 grammes per 100 ml.; serum bilirubin content, 12 mg. per 100 ml.; serum alkaline phosphatase content, 25 King-Armstrong units per 100 ml.; thymol flocculation test, negative result; zinc sulphate turbidity 4·5 units (normal 2 to 6 units). An X-ray examination of the chest gave a normal result. The results of the hydatid complement fixation test and serological tests for syphilis were negative, and no malarial parasites could be seen in the blood.

On September 25, slow transfusion with packed red cells was commenced, and despite a rate of less than five drops per minute, breathlessness and raised venous pressure ensued, necessitating rapid digitalization. Over a period of 48 hours it was possible to infuse, in all, 1 litre of packed red cells. Prednisone therapy was commenced on September 26, in a dose of 90 mg. in the first 24 hours, 60 mg. in the following 24 hours and thereafter 30 mg. per day in three divided doses.

Subjective and objective improvement were obvious by September 27; the temperature had fallen to normal, appetite was returning and the patient was able, without any fatigue, to carry on a lengthy discussion of the sequence of events in his illness; it was on this day that methyl chloride was first incriminated, and on the same day the Government Analyst reported that the 24-hour urinary heavy metal excretions were within normal limits. Improvement was progressive and rapid from the time of initiation of prednisone therapy, and the progressive alteration in the results of biochemical tests may be seen in Table I. Proteinuria continued until October 7, and the results of tests for bile pigments in the urine were positive until October 8.

Aspiration liver biopsy was performed on October 23, and was reported on by Dr. E. F. Thomson as showing "necrosis with regeneration, mainly in the centres of the lobules, and early reticulin formation". The spleen remained palpable for only three weeks, but the liver edge was still just palpable at the time of his discharge from hospital. Bed rest was insisted upon until October 26, and on November 4, the patient was allowed to leave hospital with instructions regarding rest. Prednisone dosage was reduced very gradually, so that when he was readmitted for further assessment on February 21, 1958, he was taking 10 mg. per day. At this time he looked healthy, and complained only of a dull epigastric ache and mild breathlessness on exertion. There were no abnormal physical findings, examination of the urine revealed no abnormality and his weight was 144 lb. His hæmoglobin value was 16·6 grammes per 100 ml., and the white cell count was normal; the blood sedimentation rate was also normal, and the results of liver function tests are set out in Table I. Liver biopsy was reported as normal by Dr. V. J. McGovern, and the patient was discharged to the care of his local doctor, still taking a small dose of prednisone.

Since this time the patient has built a house for himself, and when last examined, in September, 1958, was planning a business trip to Japan.

Discussion.

This patient had worked with methyl chloride for some 18 years, but only in the six months prior to his admission to hospital had exposure been particularly heavy; this period corresponds with the duration of symptoms. Although a close study has been made, there seems to be no possibility of significant exposure to any chemicals except methyl chloride and, perhaps, caustic soda.

The usual features of the disorder have already been discussed; the only one of them present in this patient was blurring of vision. However, his major manifestations (liver damage, acute hæmolysis and, presumably, renal damage as judged by albuminuria) have all occurred before in well-documented cases.

Proof of diagnosis is not possible, as has been previously discussed, although the strong history of exposure (and its close correlation in time with the symptoms), together with a rather limited field of alternative diagnoses, must be regarded as very strong circumstantial evidence.

Liver damage was demonstrated by biopsy; hæmolysis is proven by the presence of anæmia, a positive response to Schumm's test and the leukæmoid response seen in the initial blood count; while renal damage is strongly suggested by proteinuria lasting for three weeks after the commencement of therapy. Apart from transient blurring of vision, evidence of damage to the nervous system is completely lacking, while the respiratory system seems also to have escaped.

Eighteen months after his acute episode this patient's liver was still easily palpable, although he felt perfectly well, and further observation will obviously be necessary before it can be stated with any certainty that there have been no permanent sequelæ, but at least it can be stated that in the acute stage, death would very likely have ensued had it not been for the administration of adrenal steroids.

Particular attention is drawn to the patient's complaint that the cylinders of methyl chloride which he was using were clearly marked "Non-Toxic".

Summary.

The relevant literature on methyl chloride intoxication is reviewed, and a further unusual case is presented, with evidence of liver damage, renal damage and acute hæmolysis.

Acknowledgements.

My particular thanks are due to Dr. T. M. Greenaway, Senior Honorary Physician at Royal Prince Alfred Hospital, for his permission to publish this case, and to Professor R. H. Thorp, Professor of Pharmacology, University of Sydney, for his help with the references.

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MULTIPLE ANTIBODIES IMITATING THE PRESENCE OF A PANAGGLUTININ IN THE SERUM OF A PATIENT SUFFERING FROM HÆMOLYTIC ANÆMIA.

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THE difficulty of finding compatible blood for patients with hæmolytic anæmia is well known. In many instances each sample of red cells tested reacts with the patient's serum either by some or by all methods used, giving the appearance of panagglutination.

In 1953, Weiner, Battey, Cleghorn, Marson and Meynell demonstrated that in one of their cases this "panagglutinin" was of blood-group specificity and was directed against a component of the patient's own blood type. The antibody was classified as anti-e(anti-hr"), and the patient's Rh type was CDe/CDe(R_1R_1). There was no history of transfusion or any other injection of blood. As the red cells of about 98% of white people have a corresponding factor to anti-e(anti-hr"), the presence of such an antibody may well imitate the reaction of a panagglutinin. Since then, many more examples of anti-e(anti-hr"), as well as other blood-group specific antibodies antagonistic to the patient's own cells, have been reported in cases of hæmolytic anæmia. This introduces a new aspect into blood transfusion practice in adults, for in such cases the ideal blood to give is not that which is identical with the recipient's own blood factor composition, but blood which lacks the factor or factors against which the patient has become autoimmunized.

The present report describes the characteristics of a human serum which, by its mixture of autoantibodies and isoantibodies, appeared to be of a panagglutinating nature. However, absorption and elution tests disclosed the presence of four separate antibodies which, combined, act on all human red cells with the exception of homozygous –D–, described by Race, Sanger and Selwyn (1950). This serum was referred to us by Dr. Ian Wood and Dr. Ian Mackay, of the Walter and Eliza Hall Institute of Medical Research, with the following clinical data.

Miss Bg, aged 15 years, was suffering from an acquired hæmolytic anæmia of several weeks' duration. She gave a single positive response to the L.E. cell test, and the result of the autoimmune complement fixation test with liver antigen was positive to a titre of 1:512 (Mackay and Larkin, 1958). This was regarded as supportive evidence for lupus erythematosus. However, the provisional diagnosis of lupus erythematosus has not yet been fully confirmed.

She was transfused with three pints of blood in August, 1959. The Rh types of the donors used were as follows:

1st donor: O R_1^wr (C^wDe/cde)
2nd donor: O R_1r (CDe/cde)
3rd donor: O R_2r (cDE/cde)

In January, 1960, splenectomy was contemplated, and six pints of blood were cross-matched for the operation. All were found to be incompatible. A sample of her blood was then sent to us for further investigation.

Miss Bg was Group O, R_1R_1(CDe/CDe), MNS, P+, Le(a–), Fy(a+), K+, Lu(a–), Jk(a+), Wr(a+). Her red cells gave a strong, positive direct Coombs reaction, and her serum reacted with all Group O cells against which it was tested. The cell panel was selected in such a way that the presence or absence of antibodies of the Rh, Fy, MNS, K, Jk and Le systems could be ascertained. By the use of the modified two-stage papain method described by Albrey and Simmons (1960), the reactions with all cell samples varied slightly between "+++" and "++++" positive readings. The differences became more pronounced when the indirect Coombs, albumin or P.V.P. methods were used, and the weakest reactions were obtained with cells of type R_1R_1 independent of their classification in other blood-group systems. The antibody or antibodies appeared to be associated with the Rh system, so attempts were made to elucidate their specificity by absorption with, and elution from, cells of varying Rh pattern.

The cells used for absorption were papainized by the thioglycollic acid-papain method as described by Pond (1960), which is particularly suitable for bulk papainization. The absorbed sera were tested by the modified two-stage papain method which utilizes a stable activated papain solution of approximate pH 3·0.

The results of the absorptions and the agglutination strengths of cell samples of various Rh types, together with the probable reacting antigens, are shown in Table I.

TABLE I.

Serum Bg Tested by the Modified Two-Stage Papain Method for Antibodies after Absorption with Papainized Cells of Different Rh Types.

Cells Used for Antibody Detection.	Reactions of Unabsorbed Bg Serum.	Cells Used for Absorption of Serum Bg.			
		rr.	R_1R_1.	R_1^wr.	$R_1^wR_1$.
R_1^wr (C^wDe/cde)	4[1]	3 (C^w)[2]	4 (C^we)	2 (e)	4 (e)
$R_1^wR_1$ (C^wDe/CDe)	4	3 (C^w)	3 (C^w)	–	–
R_1r (CDe/cde)	4	Tr (c)	4 (c)	2 (c)	4 (c)
R_1r (CDe/cde)	4	–	4 (c)	1 (c)	4 (c)
R_2r (cDE/cde)	4	4 (E)	4 (Ec)	4 (E)	4 (Ec)
R_2R_2 (cDE/cDE)	4	4 (E)	4 (Ec)	4 (E)	4 (Ec)
rr (cde/cde)	4	1 (c)	4 (c)	3 (c)	4 (c)
R_1R_1 (CDe/CDe)	3	–	–	–	–
R_1R_1 (CDe/CDe)	3	–	–	–	–
Bg Cells: R_1R_1 (CDe/CDe)	3	2	2	2	2

[1] Figures indicate degrees of agglutination.

[2] Letters in parentheses show probable reacting antigen(s).

From the results in Table I, it appeared that serum Bg contained a mixture of at least anti-c, anti-E and anti-C^w antibodies, as absorption with rr cells almost completely prevented subsequent reactions with R_1r and rr cells, but left components behind which still reacted with E or C^w antigens. The absorption of anti-c by R_1^wr cells was not complete, but it was sufficient to show reductions of titre.

Absorption and elution tests confirmed the presence of these three antibodies. The elutions were carried out by the method of Weiner (1957), and the absorptions were made as shown in Table II.

None of the three antibodies anti-c, anti-E and anti-C^w, however, could be responsible for the agglutination of R_1R_1 cells by serum Bg, an agglutination which was readily prevented by absorptions with cells of Rh types R_1R_1, $R_1{}^wr$ and rr (see Table I). The reactions with R_1R_1 cells seemed to indicate the presence of anti-e as an additional antibody, despite the fact that the e antigen is one of the Rh antigens possessed by the patient. The results shown in Table II, which were

TABLE II.

Rh Reactions Obtained by the Modified Two-Stage Papain Method with Eluates from Papainized Cells of Different Rh Types after Contact with Serum Bg.

Cells Used for Testing.	Eluates from Contact Cells.			
	R_1R_1.	R_2R_2.	R_1R_1.	$R_1{}^wr$.
$R_1{}^wr$ (C^{w}De/cde)	+	+	−	-[1]
$R_1{}^wR_2$ (C^{w}De/cDE)	+	+	−	+
$R_1{}^wR_1$ (C^{w}De/CDe)	−	−	−	-
R_2R_2 (CDe/cDE)	+	+	−	+
R_1R_1 (CDe/CDe)	−	−	−	−
R_1R_1 (CDe/CDe)	−	−	−	−

[1] Tests not done, because of insufficient eluate.

obtained with eluates, failed completely to indicate anti-e activity, even though its presence seemed certain.

The serum Bg was then absorbed twice with R_2R_2 cells to remove anti-c and anti-E. By the use of the very sensitive technique of the indirect Coombs test on papainized cells, the absorbed serum was tested with R_1R_1 and R_2R_2 cells, and agglutination was obtained with R_1R_1 cells possessing the e antigen, but no agglutination was evident with R_2R_2 cells lacking the e antigen (see Table III).

TABLE III.

Rh Reactions Obtained by the Indirect Coombs Test on Papainized Cells with Serum Bg after Absorption with R_2R_2 Cells.

Test Cells.	Serum Bg Absorbed Twice with R_2R_2 Cells.
R_1R_1 (CDe/CDe)	+
R_2R_2 (cDE/cDE)	−

Finally, Miss Bg's papainized cells were incubated with her own serum at 37° C. for 60 minutes, the antibody was eluted and the eulate was tested against R_1R_1 and R_2R_2 cells, again with the use of the indirect Coombs test on papainized cells. R_1R_1 cells agglutinated, and R_2R_2 cells did not agglutinate, as before (see Table IV).

Both experiments (Tables III and IV) confirm the presence of anti-e. Although this antibody could be easily absorbed by appropriate cells, it could be demonstrated in eluates only by the most sensitive method at our disposal.

Discussion.

During recent years, acquired hæmolytic anæmia has become a serological problem as well as a hæmatological one. Dameshek and Schwartz (1938) showed that hæmolytic anæmia could be caused by a red-cell antigen-antibody linkage, by producing this disease in guinea-pigs after injecting them with anti-guinea-pig red-cell rabbit serum. Boorman, Dodd and Loutit (1946) proved the existence of such a linkage by demonstrating a direct positive Coombs reaction on the red cells of most patients suffering from this disease, and finally Weiner *et alii* (1953), completed the picture by identifying the circulating antibody as a specific blood-group antibody acting on a blood-group component of the patient's own red cells.

Three of the four antibodies in Miss Bg's serum (anti-E, anti-C^w and anti-c) were most likely produced by transfusions. She had previously received blood from three donors, a total of three pints, all of which carried the c antigen, but only one had the C^w and one the E antigen. Miss Bg appears to be a good antibody producer, thus resembling most patients suffering from lupus erythematosus, itself an autoantibody disease.

TABLE IV.

Rh Reactions Obtained by the Indirect Coombs Test on Papainized Cells with Eluate from Papainized Bg Cells.

Test Cells.	Eluate from Cells Bg after Contact with Serum Bg.
R_1R_1 (CDe/CDe)	+
R_2R_2 (cDE/cDE)	−

These three antibodies are foreign to the patient's own Rh system. The fourth, anti-e, is directed against her own cells. We do not know how it was produced. It could have been caused by the transfusions, as all three donors carried e antigen on their red cells. In keeping with other cases reported, it is more likely to be of non-blood origin. A pre-transfusion sample, which had been kept in the deep-freeze refrigerator for a considerable time, already showed panagglutination with papain-treated cells, but the amount was too small to carry out all the necessary tests for full identification.

Miss Bg, who is Kidd, Kell and Duffy positive, and therefore (physiologically) unlikely to form antibodies for these systems, has developed only antibodies belonging to the Rh family. With those four antibodies the patient's serum had to be panagglutinating, as the combined action even of only two of them, anti-E and anti-e, would affect almost all human red cells. The only strictly compatible blood for her would not be her own type R_1R_1, but the very rare type of homozygous –D– blood. This proved to be the case when Miss Bg's serum was tested against group O –D–/–D– (Eldridge) blood previously sent to us, in December, 1959, by Dr. F. H. Allen of Boston, U.S.A. This stored blood reacted normally in the various blood-group systems tested to check its agglutinability.

Summary.

The blood-group serology of a female patient suffering from acquired hæmolytic anæmia, and in whose case a provisional diagnosis of lupus erythematosus was made, is described.

Her red cells gave a direct positive Coombs reaction and her serum appeared to contain a panagglutinin. Absorption and elution tests disclosed the presence of four antibodies of the Rh system — anti-c(anti-hr′), anti-E(anti-rh″), anti-C^w(anti-rh^w) and anti-e(anti-hr″). The first three antibodies had been produced against Rh antigens not possessed by the patient, while the fourth, anti-e(anti-hr″) acted on the patient's own cells, which were of the type R_1R_1 and could be eluted from them.

It was shown that group O –D–/–D– blood was compatible with Miss Bg's serum, and only blood of this extremely rare Rh type would prove suitable for transfusion.

Acknowledgement.

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THE FREQUENCY OF RIGHT INGUINAL HERNIA AFTER APPENDICECTOMY: AN EXERCISE IN SIGNIFICANCE.

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IT is a clinical impression that right inguinal hernia is seen more frequently in patients after appendicectomy than in patients who have never had their appendix removed.

A mechanism is easy to postulate — namely, the section or stretching or ligation of the sub-costal or ilio-hypogastric nerve running medially to innervate the lower parts of the internal oblique and transversus muscles, where they provide the shutter mechanism at the internal inguinal ring and roof over the inguinal canal.

How can this clinical impression be tested by observation? It would be difficult to follow all patients after appendicectomy prospectively for many years, and similarly difficult to provide a control group representative of the population which covered the same age range and which had not had appendicectomy. However, the problem can be rearranged by finding the proportion of those patients presenting with inguinal hernia whose appendix had been removed previously.

To assess the frequency of appendicectomy through a right iliac fossa incision before the occurrence of right inguinal hernia, the records of 252 consecutive patients presenting for herniorraphy at the Hammersmith Hospital, London, were examined retrospectively.

Ninety-seven patients presented with right inguinal hernia, in 12 (12·4%) of whom the appendix had been previously removed through a right muscle-splitting incision. Eighty-one patients had left inguinal hernias, of whom seven (9·5%) had previous appendicectomies; and 74 had bilateral inguinal hernias, of whom four (5·4%) had previous appendicectomies. Are these differences likely or unlikely to have appeared by chance in samples of these sizes? That is, are these differences "significant"?

It is in this field of partial or intermittent effects from partial or intermittent causes that the clinical investigator finds difficulty in being confident in the meaning or significance of his observations. In fact, this paper arose from the inability to make any useful decision about these figures, and from the insecurity one felt in the presence of statistical jargon. Full-time research workers of necessity employ statistical techniques in the analysis of the data they accumulate, but most members of the medical profession meet only the end results as formulæ in small print. This results in either blind acceptance as an act of faith, or equally blind hostility to the unknown.

Since the practice of medicine is guided by the published work of others, it becomes a matter of individual responsibility to separate the chaff from the grain, and to decide which conclusions have been validly drawn and which rest on scanty inference. Not that the addition of statistical analysis makes a piece of investigation better or worse; but, if it is properly done, it indicates the degree of confidence we can feel in the meaning of that particular set of results.

It is, then, to clarify the usefulness of inferences drawn from small samples, that statistical methods may be used.

How can the apparent differences in these figures be tested? Investigation by statistical methods follows a certain broad procedure, although there is often some choice in the actual test used. The first step is to propose a null hypothesis. This will generally be the absence of an effect or impression in the data to be considered. In this example, the null hypothesis is that the occurrence of previous appendicectomy is independent of the side on which the hernia presents — that is, that previous appendicectomy is no more frequent before right inguinal hernias than before left or bilateral hernias. This move removes the bias that the investigator feels towards his acquired figures (for it is usually more exciting to report a positive than a negative effect).

This hypothesis now stands, and is rejected only if the data prove to be significantly inconsistent with it. The numbers of patients presenting with right, left, and bilateral hernias were, of course, different; but if the null hypothesis was true, we should expect approximately the same proportion in each group to have had a previous appendicectomy. Chance selection of the patients entering the hospital at the time of study would make these proportions vary.

The problem is to decide by how much the proportions can differ, and still be consistent with chance variations from the null hypothesis.

If probability testing shows that the difference could arise frequently by chance in sampling, we should retain our null hypothesis of no difference, however suggestive the original figures may appear on the surface.

If it is shown that the figures are unlikely to have occurred by chance selection, the null hypothesis can be rejected on the grounds that it is a less likely explanation than that of association — i.e., that the difference is "significant".

Since this rejection or acceptance is based on probability, we must indicate the degree of that probability to give some measurable reality to the term "significant".

The probability can be written thus: "This difference could have occurred by chance in selection once in a hundred (or whatever figure applies) of such series." The shorthand for this is $P = 0{\cdot}01$ — that is, the probability of chance occurrence is one in 100.

As a convention, a probability of less than one in 20 trials ($P = 0{\cdot}05$ or 5%) is called "probably significant", one in 100 ($P = 0{\cdot}01$ or 1%) is "significant", and one in 1000 ($P = 0{\cdot}001$ or 0·1%) is "highly significant". If a probability cannot be stated exactly, its limits might be, for example, $0{\cdot}05 > P > 0{\cdot}01$.

This statement of a probability figure reveals the heart of the problem. We are not constructing a "mathematical proof or rebuttal", but indicating the degree of confidence we feel in accepting or rejecting an hypothesis. Clearly risks are involved in such decisions — the probability figure gives a measure of that risk.

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Even though data are apparently unselected, or selected randomly, we can be unlucky in one of two ways. The particular figures can give a false positive with rejection of the null hypothesis, or a false negative in which the null hypothesis is retained. Of course, these errors are created by the original figures, not by the subsequent statistical assessment.

Generally only the risks of the false positive are considered, and before analysis we specify the risk we are prepared to take.

Suppose we decide on a risk of 0·05; then there is once chance in 20 that our test will lead us to reject the null hypothesis when it is true. If this risk appears too high, it can be reduced, for example, to 0·01 (that is, one in 100), but always at the expense of creating a false negative by applying too stringent a test. The risk of creating a false negative increases as the difference between the test group and the control becomes less—here the difference between right inguinal hernia and the rest in relation to previous appendicectomy. If the difference is small, there is little enough chance of detecting it, and that chance of detection decreases as we make the test more stringent. Fortunately such marginal effects are rarely of importance.

Thus the steps followed are to set up a null hypothesis, to decide upon the test to be used and to set the risk limits.

Now we are ready to analyse the data, for we have reduced them to a form for substitution of figures, and the decision follows automatically. This removes the temptation to adjust the risk, and thereby the test, to conform to the notions of the experimenter.

In the present case the test is really a test of the dependence or independence of the occurrence of previous appendicectomy on the side of the hernia.

TABLE I.
Observations.

Appendicectomy.	Hernia.			Total.
	Right.	Left.	Bilateral.	
No previous appendicectomy	85	74	70	229
Previous appendicectomy ..	12	7	4	23
Total	97	81	74	252

If the null hypothesis was true, we should expect each group to show the same proportion of previous appendicectomy as is seen in the total—that is, 23 out of 252. Of 97 patients with right inguinal hernias, we should expect 8·8 to have had previous appendicectomy (the decimal point should not concern us, for this can be thought of as the average taken over many repetitions of the experiment).

Similar expected values can be calculated for each group.

The data are now in a form in which we can use the χ^2 test. In each group in the table, the difference between the observed value and the expected value is squared and divided by the expected value. The sum of these terms $\frac{(\text{observed number}-\text{expected number})^2}{\text{expected number}}$ has approximately the distribution known as χ^2.

$$\text{The sum} = \frac{(85-88{\cdot}2)^2}{88{\cdot}2} + \frac{(12-8{\cdot}8)^2}{8{\cdot}8} + \frac{(74-73{\cdot}6)^2}{73{\cdot}6} + \frac{(7-7{\cdot}4)^2}{7{\cdot}4} + \frac{(70-67{\cdot}2)^2}{67{\cdot}2} + \frac{(4-6{\cdot}8)^2}{6{\cdot}8}$$

$$= 2{\cdot}6 \text{ approximately.}$$

This sum may be called χ^2, and the probability that these differences from the expected numbers occurred by chance is found by consulting probability tables for levels of χ^2, taking into account the number of independent subgroups which contributed to its total. The number of groups or "degrees of freedom" is the number

TABLE II.
Expected Numbers.

Appendicectomy.	Hernia.			Total.
	Right.	Left.	Bilateral.	
No previous appendicectomy	88·2	73·6	67·2	229
Previous appendicectomy ..	8·8	7·4	6·8	23
Total	97	81	74	252

of classes whose frequency could be assigned arbitrarily—that is, the number of independently variable groups. Of the six numbers entering into this calculation, only two required separate measurement, the others being derived as inverse proportions or by subtraction from the totals. The number (n) of degrees of freedom can also be found from the formula $n = (c-1)(r-1)$, where c is the number of columns (excluding the totals column), and r is the number of rows.

On consulting probability tables, a value of $\chi^2 = 2{\cdot}6$ where $n = 2$ is seen to give a probability (P) between 0·2 and 0·3 that the differences were due to chance. To reach our arbitrary 0·05 (1 in 20) level of significance, χ^2 would need to be greater than 6.

This value of $\chi^2 = 2{\cdot}6$ is consistent with the hypothesis of no difference. In more formal words, the data were consistent with the hypothesis that the incidence of appendicectomy before repair of right inguinal hernia was the same as the incidence of appendicectomy before repair of left-sided and bilateral hernias. Any difference was too small to be detected by this experiment.

Conclusion.

In this sample, appendicectomy before right inguinal herniorraphy (12·4%) is more frequent than appendicectomy before left and bilateral herniorraphy (11 in 155, 7·1%), and this difference is sufficiently wide to suggest some association between appendicectomy and right inguinal hernia. However, the arrangement of the data to remove bias, and the application of a simple statistical test, show that such a difference in frequency could easily have arisen by chance.

Summary.

1. The frequency of appendicectomy before inguinal herniorraphy has been noted in 252 patients.

2. The χ^2 test of significance has been demonstrated on these data.

Acknowledgements.

Professor Ian Aird broached this problem, and kindly gave permission for these observations to be published. Dr. G. Gregory, of the Department of Statistics, University of Melbourne, gave extremely valuable and careful criticism of this paper.

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Reports of Cases.

CHRONIC IDIOPATHIC JAUNDICE WITH HEPATIC PIGMENTATION (DUBIN-JOHNSON SYNDROME).

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AND
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IN 1954, Dubin and Johnson, and Sprinz and Nelson described a syndrome of jaundice associated with pigment deposition in the liver cells, and in 1958, Dubin was able to collect some 50 cases of the syndrome. Since then a number of cases have been described, the first Australian one in 1959 by Taft and Earle. A further case occurring in a female, in which a definitive diagnosis was made by needle biopsy of the liver, is reported.

Clinical Record.

A married, nulliparious woman, aged 47 years, was admitted to the Brisbane Hospital in January, 1959, after an episode of epigastric pain and melæna for which no cause was found. She also gave a history of episodic jaundice, associated with mild ill health, extending over 21 years, and on this admission to hospital, the serum bilirubin level was slightly elevated. She was readmitted for investigation of her jaundice in March, 1959, when she provided a history of recurrent jaundice from the age of 26 years. Mostly the episodes of jaundice were mild, occurring during the early summer months and being preceded by symptoms suggesting an upper respiratory tract infection. The episodes were accompanied by nausea, abdominal discomfort and a sensation of distension, with a distinct unsteadiness on standing. Sometimes diarrhœa and vomiting were present. Mental depression frequently accompanied or followed the attacks, and in recent years had become a more prominent feature, lasting from two to six weeks.

The patient was born in Brisbane in 1912, but since 1927 she has lived for varying periods in Ceylon and England, as well as in Australia. Past illnesses have included diphtheria and scarlet fever during childhood, and recurrent malaria during the years from 1928 to 1934. In 1941 she was informed that her gall-bladder functioned abnormally on cholecystography. In 1947 the result of a hippuric acid excretion test was stated to be normal and that of a cholecystogram abnormal; on this occasion an abdominal exploration revealed a normal extrahepatic biliary system, and an hepatic biopsy was performed, but no adequate diagnosis was made. Unfortunately, neither the histological report on the liver tissue nor the biopsy specimen itself is now available. No family history of jaundice was known; the patient's mother died from a malignant disease of the stomach at the age of 32 years, and her father from "acute heart failure" at the age of 35 years. One sibling was stillborn and one died in early infancy from unknown causes, but her surviving brother, who lived in Ceylon, was apparently well some years ago.

When examined in March, 1959, the patient was thin, with minor scleral icterus. The liver was palpated two fingers' breadth below the right costal margin, in the anterior axillary line, and its edge seemed firmer than normal; the spleen was not palpable. There was no further evidence of hepatic or other disease.

The serum bilirubin level was 1·2 mg. and 1·3 mg. per 100 millilitres on two occasion, and the Van den Bergh reaction was biphasic; the fæces were normal in colour and the urine did not contain bile pigments or increased amounts of urobilinogen. The serum alkaline phosphatase level was 3·9 and 4·0 King-Armstrong units per 100 ml. on two occasions, the serum cholesterol level was 212 mg. per 100 ml. and the total serum protein level was between 6·9 and 7·2 grammes per 100 ml., with a normal pattern on paper electrophoresis. Readings of the thymol turbidity test varied from one to two units, and the cephalin cholesterol flocculation test gave a negative result; the one-stage prothrombin time was not prolonged; 6% bromsulphalein solution was retained 45 minutes after a standard dose. Cholescystography indicated poor dye concentration in the gall-bladder, but no other abnormality. Hæmatological investigations revealed no significant abnormality. The hæmoglobin value was 13·4 grammes per 100 ml.; reticulocytes numbered 0·2% to 0·5%; the appearances of the blood cells in a stained smear were normal; the hæmatocrit reading was 40%; the M.C.V. was 95 c.μ., the M.C.H. was 31 $\gamma\gamma$ and the M.C.H.C. was 32%; the direct Coombs' test and Schumm's test both gave negative results; the osmotic fragility of the red cells was normal; serological tests for syphilis gave negative results; and the erythrocyte sedimentation rate was not increased.

Percutaneous needle biopsy of the liver was performed, and the histological findings were as follows:

> Sections show no evidence of cirrhosis or of inflammation. The liver cells appear normal, and there are no bile thrombi, but there is a gross amount of greenish-brown, granular pigment present orientated about the canaliculi and filling an occasional liver cell. It is present also in Kupffer cells. The granules vary from 5 microns to less than 1 micron in diameter and the larger vary slightly from the smaller in their staining reactions. [See Figures I and II.[1]] In paraffin sections of formalin-fixed tissue, the granules were isotropic to polarised light and were acid fast. Gmelin's test, the Prussian blue, periodic acid-Schiff and iron hæmatoxylin reactions were all negative; the Nile Blue sulphate and diamine silver reduction reactions were positive; Stein's test was negative except for a few large granules and Schmorl's reaction was positive except for a few large granules; bleaching with hydrogen peroxide was slow, and incomplete in the large granules, and Mallory's basic-fuchsin stained a few large granules. The appearances are consistent with chronic idiopathic jaundice with pigment granules in the liver.

Discussion.

The clinical history, physical and biochemical findings and hepatic histology in this case are characteristic of those found in chronic idiopathic jaundice with granular hepatic pigmentation. The only certain method of diagnosis is by histological examination of liver tissue; consequently, hepatic biopsy is the most important single procedure. The Van den Bergh reaction has been reported as biphasic in most cases of the Dubin-Johnson syndrome, as it was in this one, suggesting that some of the bilirubin in serum is conjugated. This has been confirmed by Burka (1960) who, in a similar case, showed that 62% of the serum bilirubin was direct-reacting, 22% being diglucuronide and 78% monoglucuronide. If a biphasic Van den Bergh reaction is present, Gilbert's disease (familial non-hæmolytic jaundice) is excluded, since in that disorder the serum bilirubin level is raised because of the indirect-reacting (unconjugated) bilirubin. The long history, and the minimal systemic disturbance, normal hepatic function other than the elevation of the serum bilirubin level, and normal hæmatological findings, virtually exclude hepatocellular and obstructive jaundice and hæmolytic processes.

Since the previous histological sections of this patient's liver are no longer available, it has not been possible to determine whether pigment deposition increased over the years as in the case reported by Taft and Earle (1959).

Summary.

A further case of the Dubin-Johnson syndrome in a female is described.

Acknowledgements.

We wish to thank Professor N. Sutton for referring this patient to us for investigation, Dr. Margaret Mead for the detailed histological report, and Dr. A. Knyvett for permission to use hospital records.

[1] For Figures I and II see art-paper supplement.

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Legends to Illustrations.

FIGURE I.—Section of liver (hæmatoxylin and eosin stain); low power of the microscope.

FIGURE II.—High power view of same section.

ARTERIOSCLEROSIS OF THE ARTERIES OF THE STOMACH.

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APART from venous bleeding in portal hypertension, it is rare for primary intrinsic vascular lesions to be responsible for gastro-intestinal hæmorrhage. In the majority of instances the offending vessel is secondarily involved by extrinsic factors usually of an ulcerating type, either peptic or neoplastic. The occurrence of two cases of bleeding from gastric vessels, in each of which the essential defect was primarily vascular, was therefore considered worthy of note. In one the artery was involved in its extragastric course, giving rise to extensive hæmoperitoneum. In the other the site was submucosal, leading to hæmorrhage within the cavity of the stomach.

Case I.

An invalid pensioner, aged 64 years, had suffered indifferent health for years, and had a past history of arteriosclerosis, hypertension, congestive cardiac failure and spinal deformity. Thirty-six hours prior to his admission to hospital he noticed the gradual onset of epigastric pain, at first not severe and chiefly intermittent in character. This was attended by anorexia and later in the day by vomiting on three occasions. Later a sudden increase occurred in the pain, which was still colicky, but now very severe, and maximal in the epigastrium, with radiation down the left side of the abdomen. Vomiting was repeated on his admission to hospital, and one-quarter of a grain of morphine did not completely relieve the pain. There had been no weight loss, and there was no past history of indigestion. Gross dyspnœa of effort, even on walking 50 yards, had been present for years.

Examination of the patient showed him to be a pale, thin, ill-looking elderly man. The blood pressure was 170/105 mm. of mercury. The radial pulse was of good volume, the vessel being thickened and tortuous. A gross depression of the lower ribs and sternum due to the long-standing deformity of pectus excavatum was evident. A harsh, systolic murmur was audible at the apex of the heart. Examination of the abdomen showed considerable rigidity over its whole extent, most pronounced in the epigastrium, which was also the site of maximum tenderness. Tenderness was also evident down the left side into the iliac fossa. There was no diminution of liver dullness and no obvious abdominal distension.

A presumptive diagnosis of perforation of a peptic ulcer or gastric carcinoma was made. The patient was prepared for operation after a short period of resuscitation and intravenous therapy. Laparotomy revealed a large quantity of blood free in the general peritoneal cavity and lesser sac, which were explored in an endeavour to locate the source of bleeding. A diligent search found the left gastric artery to be only partly pulsating, with blood oozing freely at a point midway along the lesser curvature at the site of anastomosis with the right gastric artery. The vessel in question and the other arteries of the stomach felt very much firmer in consistency than normal, rather like strands of whip-cord, and exhibited poor pulsation. This condition prevailed along the whole of the course of the left gastric artery to within 1·5 cm. of its origin from the cœliac axis, where its normal calibre sharply returned and pulsation could be felt by the palpating finger. Ligation in continuity was effected near its origin and again on either side, but well clear of the oozing point. The intervening segment of about 2·5 cm., which was enmeshed in subserous hæmatoma and œdema, was excised. The stomach appeared somewhat congested, with cyanosis, but showed peristaltic activity and responded to stimulation, and was therefore considered viable. The other abdominal viscera and their arteries were normal in appearance, although atheromatous plaques could be felt in the lower abdominal aorta. The abdomen was closed wthout drainage.

The patient remained in a weak, shocked state despite transfusions, and died some 24 hours later. A post-mortem examination was not obtained.

Case II.

A man, aged 66 years, had suffered a coronary occlusion four years previously, and was subject to attacks of pain of an anginal type. His present illness had commenced two days prior to his admission to hospital, with epigastric discomfort and later pain which culminated in severe hæmatemesis. He was examined at the request of his physician because of continued bleeding unresponsive to conservative measures.

Laparotomy revealed distension of the stomach, from which, after it had been opened, was removed a pint and a half of clotted and fluid blood. No lesion could be seen or felt on the outer surface; but after repeated lavage to remove adherent clot and mucus, a tiny, mammillated projection was palpable from within the lumen, high on the posterior wall of the fundus above the level of the cardia. This proved to be a dilated and indurated segment of a submucosal vessel, which at the site of the rupture was partly plugged by protruding thrombus. The surrounding mucosa was normal, without erosion or ulceration. With difficulty, on account of the depth and inaccessibility of the lesion, the vessel was underrun by a silk suture, and the gastrotomy incision was closed in layers. Wound sepsis delayed convalescence, but ultimately the patient's recovery was complete. He was discharged from hospital well, and had no further gastric disturbances.

Discussion.

Review of the literature reveals the relative infrequency of lesions of this type. Taylor in 1945 discussed ruptures of aneurysms of visceral arteries, pointing out that they might discharge either into the lumen or intraperitoneally. Both he and Green and Powers (1931) gave reports of findings identical with those in Case I. The sites of rupture were similar, in that they occurred at the anastomotic junction between the right and left gastric vessels.

Charles Morton in 1938 summarized the literature, to find 12 cases of spontaneous rupture of gastro-intestinal vessels. In six instances the rupture involved the left gastric artery, in three the superior mesenteric artery, in two the right gastric artery, and in one the gastro-duodenal artery.

The initiating disturbance which precipitates the rupture may be developmental, degenerative, inflammatory or syphilitic. Crile and Newell (1940) remark on the rarity of spontaneous rupture of visceral vessels, in contrast to the prevalence of degenerative vascular disease elsewhere. They reported 10 cases of intra-

abdominal apoplexy, in eight of which operation was undertaken with only four survivals.

In the second case reported here, the cause of hæmorrhage proved to be a primary lesion originating in a gastric vessel. In this instance a submucosal artery of some considerable calibre was involved, with the loss of a large quantity of blood into the cavity of the stomach.

Erosion of vessels by the action of peptic digestion is of common occurrence, but other causes of rupture are rare. Monaci lists these as arteriosclerosis, developmental malformations, mycotic aneurysms, syphilitic involvement of arteries and trauma. Whilst arteriosclerosis was responsible for the rupture in Case I, the cause was not conclusively determined in Case II. However, in the complete absence of peptic digestion around the ruptured vessel, a degenerative or mycotic process appeared as most likely.

An interesting report comes from Frank (1946) of a man who, at the age of 16 years, experienced hæmatemesis followed by episodes of severe intermittent gastric bleeding over a period of 30 years, the cause of which was not directly established during life. After his death from pulmonary tuberculosis, autopsy revealed advanced arteriosclerosis of the submucosal branches of the gastric arteries. The affected arteries were of extraordinarily large size for submucosal vessels. Frank reviewed the literature, and concluded that there were few reported cases of severe gastric arteriosclerosis; the gastric arteries were less frequently and less severely involved than vessels elsewhere. He also stated that all patients recorded in the literature as having severe arteriosclerosis of the gastric submucosal arteries had manifested clinically unexplained repeated gastric hæmorrhages from adolescence to senescence. Frank analysed the gastric vessels in 100 autopsy specimens, finding only two instances of moderately severe arteriosclerosis, with no previous clinical history of gastric disturbance in either case. In 35 cases in the series, minimal to mild sclerosis was found in the smaller submucosal ramifications of the gastric arteries. Frank considers that bleeding may be due either to erosion of these large arteries abutting the submucosa, or to aneurysmal rupture on the basis of an arteriosclerotic process.

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Reviews.

Gastric Cytology: Principles, Methods and Results. By Rudolf Otto Karl Schade, M.D. (Dunelm), M.D. (Tubingen), L.R.C.P., M.R.C.S.; 1960. London: Edward Arnold Ltd. 9¾" × 7¼", pp. 92 with 85 illustrations. Price: 59s. 3d.

IT is a simple task to diagnose cancer of the stomach when the lesion is far advanced and perhaps beyond surgical cure, but the early diagnosis presents a problem to physician, radiologist and surgeon. Even the gastroscopist may be in a dilemma. The study of gastric cytology in material obtained by gastric lavage and centrifugation is making a further contribution to accurate diagnosis, and this monograph by Rudolf O. K. Schade, of the University of Durham, England, is a timely contribution to our literature. Dr. Schade first traces the development of modern techniques since the early work on gastric washings by Rosenbach in 1882, and the classical researches in general exfoliative cytology by Papanicolaou and Stockard in 1917. Then follows a description of the modern techniques for collecting the washings and preparing them for microscopic examination and photography. He wisely emphasizes the problem of distinguishing cancer cells from inflammatory cells and epithelial cells arriving from the upper reaches of the gastro-intestinal and bronchial tracts. This demands great skill and patience—it is a time-consuming and somewhat monotonous procedure, but improved techniques may lessen the burden of the pathologist. Dr. Schade's descriptions of the various cells are illustrated by 85 black-and-white photographs; it is sad that publication costs have precluded the use of colour. After discussing chronic gastritis and the gastric atrophy of pernicious anæmia, he describes true cancer, paying special attention to an early form, "surface cancer", in which there is surface spread before tumour formation is apparent to inspection or radiology. Surface cancer is readily observed by "cytodiagnosis". Dr. Schade concludes by recording his experience in lucid tables. From 1954 to 1958, gastric washings were examined from 282 patients later proved at laparotomy to have cancer, and 97·6% of the diagnoses were accurate; in only six cases was cancer falsely excluded, the remaining 24 providing unsuitable material. Of 276 cases in which a gastric lesion was proved at laparotomy not to be cancer, the gastric washings were correctly diagnosed as being non-cancerous in 94·8%; however, in 13 cases a false positive diagnosis of cancer was made. This emphasizes that positive results should be viewed with caution, and that a final diagnosis should be made only by laparotomy and skilled histological examination. These findings also call for further research in this field—improved staining techniques to identify cancer cells and improved methods of viewing them by microscopy and photography.

Dr. Schade has done well to stimulate our interest in this intriguing technique, which has made a valuable contribution to the detection and cure of cancer. The larger Australian hospitals will consider it their duty to use these techniques in the years to come, and research workers will strive to increase the efficiency of cytodiagnosis. Dr. Schade's book will be of interest to all who wish to reduce the ravages of gastric cancer.

Medical X-Ray Technique: Principles and Applications. By G. J. Van Der Plaats; 1959. Eindhoven, Holland: Philips Technical Library. Sydney: Philips Electrical Industries Pty. Ltd. 9" × 6", pp. 492, with 213 illustrations. Price: £3 10s.

THIS book, of Continental origin, is a useful addition to the small range available on similar subjects from the United States and England. Its contents do not suffer in quality through translation to English. (It is noted in the front of the book that it has been published in French, German and Spanish as well.) Its purpose, as stated in the preface, is to provide "a text book for all radiographers and all those who are interested in medical radiology". Radiographic positioning and centering have not been included; the scope of the book covers the history, physics, mathematics and chemistry necessary for the understanding of the various sections of radiology. The presentation employs two types of print, the smaller being reserved for detail in excess of that required by the new student.

There are well-presented sections on the origins of X rays, their properties and their production in X-ray tubes. A very brief discussion of the transformer and valves is included; but, as is stated in the preface, "a certain amount of knowledge and mathematics and physics" has been assumed.

A relatively lengthy consideration of the formation of the X-ray image includes discussion of image recording with its post-war advances, and of the various qualities and defects in the recorded image (perceptibility of detail, definition and unsharpness, contrast, etc.). The qualities of fluoroscopic and intensifying screens and radiographic films are dealt with next, and then follows a good working account of the dark-room. Chapters are included on special radiographic techniques, and ancillary apparatus is discussed (Bucky tables, tomographs, serial changers, etc.).

In the therapy section which follows, the biological effects of radiation, dosimetry, the dosage of X rays, superficial, deep and contact therapy, and therapy with radioactive elements are discussed. Finally, radiation hazards and protective measures are mentioned briefly.

Throughout the book, numerous diagrams, tables and X-ray reproductions are used to good advantage. The book is a useful addition to the small range available to trainee radiographers.

The Ear, Nose and Throat Diseases of Children. By J. F. Birrell, M.D., F.R.C.S.E.; 1960. London: Cassel and Company Ltd. 9½" × 5½", pp. 392, with many illustrations. Price: 72s.

IN his preface the author states that this book has been written as an adjunct to the standard textbooks, which have a tendency to approach the specialty from the adult viewpoint. Embryology and developmental anatomy are therefore the main considerations in the sections on anatomy and physiology. Details of operative techniques are not given, because they can be read in standard textbooks. Theories on controversial subjects are fairly evaluated as they apply to children.

Sound, practical common sense is the keynote of the clinical material, with emphasis always on the commonest conditions. Nowhere is this better exemplified than in the chapter of 32 pages on the tonsil and adenoid operation, a most thorough exposition of every conceivable aspect before, during and after the operation. In the chapters on middle-ear and mastoid infections the author favours the term "oto-mastoiditis" throughout, because he holds that they are too often divorced from each other in most textbooks. Acute oto-mastoiditis is considered in separate chapters for infants and for children, and the type associated with gastro-enteritis is considered as a separate problem again.

The methods of investigation and treatment epitomize those developed at the Royal Hospital for Sick Children in Edinburgh as a result of long experience. As such, they conform with those in most Australian clinics with remarkably few exceptions. Readers will find well-reasoned answers to many perplexing problems—for example, the place of surgery in sinus infections, breathing exercises, the role of nasal allergy, deafness and its recognition, idiopathic hæmotympanum, acute laryngo-tracheo-bronchitis, tracheostomy, etc. The references at the end of each chapter are particularly helpful, because much of the literature is scattered through different pædiatric, general medical and oto-laryngological journals.

This is a book which we can thoroughly recommend. Those who are concerned with the care of children will also appreciate its worth.

Visual Aids in Cardiologic Diagnosis and Treatment. Edited by Arthur M. Master, M.D., and Ephraim Donoso, M.D.; 1960. New York and London: Grune & Stratton, Inc. 10" × 6¾", pp. 224, with illustrations. Price: $10.00.

THIS book is a compendium is based on transcripitions of the proceedings of the Silver Anniversary Meeting of the American College of Chest Physicians in 1959, to which other topics have been added. The majority of the contributors are members of the staff of the Mount Sinai Hospital, New York. It makes no claim to completeness of either material or bibliography, and is essentially the exposition of the views of one group of workers. The editors believe that in cardiology "the main responsibility of the physician today is to know what can be done and what promises to be done. He must define with precision the pathologic anatomy of a given lesion and be able to assess the extent to which that lesion has altered the cardiovascular hemodynamics". To this end, this book should be a valuable guide to all physicians.

In the past two decades, the use of machines providing visual displays of various types has influenced cardiology in three ways. First, they have made possible greater understanding of anatomical aberrations and cardio-vascular physiology; secondly, they have enabled more accurate diagnosis of an abnormality to be made; and thirdly, they have led to the surgical correction of an increasing number of diseases of the heart. An appreciation of their capabilities and limitations is therefore important.

After an introductory chapter entitled "Machine and Physician in the Present and Future of Heart Disease", the anatomy of the surgically correctible congenital cardiac malformations is briefly but clearly stated. Then follow discussions on cardiac sounds, their recording and interpretation. This section includes intracardiac phonocardiography. The electrical activity of the heart is largely treated by vectorcardiographic methods. Cardiac catheterization, dye-dilution techniques and various forms of angiocardiography are dealt with at some length. Finally, the problems of the anæsthetist and surgeon are examined. In this section, details of monitoring the patient during operation are described. (At the Mount Sinai Hospital, electrocardiograms, phonocardiograms, vascular pressures, body temperature and the electroencephalogram are continuously monitored.) Stress is laid on the need for team-work. "It does not suffice for each member of the team to know his part completely. There must be . . . a smoothly working unit." The excellence of illustration of the book by photographs and drawings is unfortunately spoilt by a few of poor quality.

The editors are to be congratulated on assembling together in a reasonable space a series of articles which clearly set out the views held at this well-known centre.

Surgery in the Aged. Edited by Frank Glenn, M.D., S. W. Moore, M.D., and John M. Beal, M.D.; 1960. New York, Toronto and London; McGraw-Hill Book Company, Inc. 10" × 7", pp. 548, with illustrations. Price: $17.50.

The surgical staff at the New York Hospital, Cornell Medical Center, have combined to present a survey of surgery in the aged, a period in which all would agree that judgement is critical and morbidity is at its highest. This volume commences with a review of the basic considerations, such as metabolic response, infections, post-operative care, anæsthesia and pre-operative assessment. Following this introduction are 20 chapters grouped under diseases of the thorax, heart and vessels, intestine, genito-urinary system and biliary systems, together with the surgery of special systems, trauma and reconstruction.

As with most composite volumes, the writing and balance are often uneven, and much of the text seems not specifically concerned with problems of the aged. For example, the chapter on hernia begins with an historical introduction, which lists several methods of treatment in use from the Middle Ages to the introduction of antiseptic surgery. This is followed by the definition of a hernia, the anatomy of the inguinal and femoral canals, ætiology, incidence and treatment; at the end of a lengthy chapter, the only conclusions reached are that there is more fat and laxity in the hernial structures of the elderly, and that associated conditions, such as hypertension and chronic bronchitis, are prevalent. This tendency runs through most, if not all, sections of the book. If, indeed, it is designed to deal with the very real change of emphasis seen in the surgery of the elderly, the text could be with advantage drastically edited, and would gain in both precision and relevance.

Neurology Simplified: A Practical Approach to the Early Diagnosis and Treatment of Neurologic Diseases Written Especially for General Practitioners and Students. By David Joseph Lafia, M.D.; 1960. Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 9" × 5¾", pp. 196, with illustrations. Price: 54s. (English).

THIS little book begins with an important preface, in which the author explains "why this book was written". It cannot be regarded as a textbook of neurology, but while textbooks of neurology will rest unopened upon the bookshelves, this little book will render constant and useful service to many doctors whose work in this field will be all the better for it. It is a practical book, a workshop book, for those doctors who are not professional neurologists. Our impression on reading it was that it dealt with the subject inadequately, but we soon realized that it was written for a purpose, and it has been very well done, even brilliantly. One must not contrast the amount of information given here with that which could be given about neurology, but instead with that which is ordinarily understood of neurology by most doctors, and so realize how much this book has to offer them. By covering in a simple and brief way almost the whole breadth of neurology, it enables the reader to appreciate the scope of the subject, and in many cases it will whet his appetite to study further. A book which does this must be regarded as a very valuable book indeed.

It is unfair to comment unduly upon omissions from such a book as this, but we would suggest that as its purpose is that of a practical primer, it should not omit any broad principle. Thus, as the reader would complete this book without seeing much reference to the potency of his psychological factors in these cases, it might be as well to stress this a little more in later editions.

The most important feature is that this book is practical rather than theoretical, in a field in which most of the

literature is theoretical, and that its practical presentation is in the modern manner. Neurological house surgeons should be required to read this book at the beginning of their terms of duty.

Cerebral Palsy and Related Disorders: A Developmental Approach to Dysfunction. By Eric Denhoff, M.D., and Isabel Pick Robinhault, Ph.D.; 1960. New York, Toronto and London: McGraw-Hill Book Company, Inc. 9″ x 6″, pp. 432. Price: $12.00.

THIS is a "solid" book, and one which will interest and help those many people—doctors, nurses and members of all ancillary disciplines—who care for the physically handicapped child. For, although concerned in the main with cerebral palsy, the canvas of the book is enlarged to depict the broader syndrome of cerebral dysfunction—of which the authors consider cerebral palsy to be only one aspect. They hold that this concept, together with the new developmental approach to diagnosis and treatment, "has brought about a complete revision of thought as well as therapy".

Perhaps the claims for originality in this "developmental" approach go beyond the bounds of actuality; but in this book the reasoning and the *modus operandi* are set out in a clear and practical system. The brain of a child is a highly complex piece of mechanism which is growing and developing; therefore, the effects of "injury"—in the widest sense of that term—can be modified or even reversed during this active period of growth and development. No aspect of cerebral dysfunction is static, and there are "unplumbed resources in every individual". Every effort must be made to develop these to their maximum.

The work of the well-known Meeting Street School Children's Rehabilitation Centre, Providence, Rhode Island, figures largely in this book and thus enhances its practical value. The case histories are well chosen. They illustrate the varied problems of those many persons affected by cerebral palsy. What we are slowly learning is that the associated sensory and psychological disturbances play an important part in cerebral palsy, and that the picture goes well beyond that of a localized neuro-muscular disorder. Perhaps for too long the "spastic" child was regarded mainly as an orthopædic problem; but we must hold the balance so that he does not become regarded mainly as a problem for the psychiatric clinic. Teamwork of all the disciplines is essential, and this book is a thoughtful and practical manual for all members of the team. The bibliography is very complete.

Two misprints were noted. On page 119 (line 7) the ratio should read 10:7 and not 1:7, and on page 289 (line 28) the pedantic would spell "occurring" with two r's.

Chemotherapy in Emotional Disorders: The Psychotherapeutic Use of Somatic Treatments. By Frederic F. Flach, M.D., F.A.P.A., and Peter F. Regan III, M.D., F.A.P.A.; 1960. New York, Toronto, London: McGraw-Hill Book Company Inc. 9″ x 6″, pp. 328. Price: $10.00.

THIS book is designed as a guide to the application of physical treatments and their integration with psychotherapy. The first part, "The Evaluation of the Patient", includes a brief description of personality, the psychological aspects of various periods in the life of the individual and psychiatric syndromes. The authors call special attention to the "devastating" effects of sexual unrest. In the second part, the chemical constitutions and clinical effects of some eighty sedative, tranquillizing and stimulant substances are reviewed in some detail. The authors present no statistics, but include a few illustrative case reports. They advise that chlorpromazine, regarded as the most valuable of the phenothiazines, should not be prescribed before hepatic function has been shown to be intact after clinical and laboratory investigation. In the treatment of overdosage with barbiturates there is no mention of bemegride, but the parenteral use of picrotoxin is recommended if respirations become very slow. A special point is made about timing in the administration of electro-convulsive therapy, which, in the opinion of the authors, should be reserved for the stable phase in depressive illnesses; but they might have stated more clearly that it may still be desirable to give a few electro-convulsive treatments in the early stages, provided that it is recognized that the effects may be partial and temporary. In the section on hormone therapy there is no reference to the use of œstrogens for the reduction of libido in the male. The third part, "Integration of Treatments", covers the treatment of psychoneurotic, schizophrenic, affective, paranoid and organic reactions, and is followed by a useful glossary of trade names.

This book contains little to inform the psychiatrist who has filed for reference the manufacturers' literature, while the general physician who proposes to include psychiatry in his practice will hardly glean enough to enable him to make a sound diagnosis.

Fundamentals of Nerve Blocking. By Vincent J. Collins, M.S., M.D., with the assistance of Emery Andrew Rovenstine, M.D.; 1960. Philadelphia: Lea & Febiger; Sydney: Angus & Robertson Ltd. 9½″ x 5½″, pp. 356 with 144 illustrations. Price: £5 4s. 6d.

HERE is a book which gathers together most of the indications and techniques for the alleviation of pain by suitable nerve blocking, and with extensive bibliographies at the end of each chapter, it provides a very useful introduction to this subject.

Control of pain comes more and more into the sphere of the specialist anæsthetist. The author is an anæsthetist, and also an expert on the subject on which he writes, having gained experience in a medical centre which has devoted much time and thought, for many years, to this particularly humanitarian branch of medicine. In the average hospital pain relief by injection is a haphazard procedure, a little being done here and there by orthopædic surgeons in their own limited field, or by general surgeons for special conditions like trigeminal neuralgia, and by few other medical practitioners. It seems logical that, as this branch of therapy is so wide, it would be done better by practitioners specializing in it, be they anæsthetists or others. From reading, one can see that in a hospital of any size the work could fully occupy a specialist with a team.

The book is well printed, and the illustrations are clear. The author is to be congratulated on his easy style. Part A tells of the surgical and medical conditions in which pain may be relieved by local injections of analgesic solutions or sclerosing fluids. Part B shows how to inject, and deals with possible complications—a much-needed knowledge, as patients are very litigation-minded in these days. Naturally, this book is not perfect. The principal errors seem to be typographical, and these are numerous. Errors in spelling do not usually cause much confusion, but they shake one's confidence in the accuracy of figures, which may be wrongly printed, with serious consequences. Thus, on page 52, concerning lidocaine, in the last paragraph it is stated that "duration of anæsthesia with 10% solution is about 1½ to 2 times longer than procaine". This refers to local analgesia produced by injection, and could be very dangerous.

A Textbook of Histology: Functional Significance of Cells and Intercellular Substances. By John C. Finerty and E. V. Cowdry; fifth edition; 1960. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson Ltd. 10″ x 6½″, pp. 574, with 502 illustrations. Price: £6 1s.

COWDRY'S "Textbook of Histology", of which this is the fifth edition, has been rewritten since its author's death by J. C. Finerty. Whereas in the fourth edition a description of the blood vascular system and endocrine organs preceded that of other organs and tissues, including supporting tissues and muscle, the present author has rearranged the chapters to conform more with the conventional sequence of teaching topics. The book has been brought up to date by the addition of a large number of electron micrographs and corresponding discussions of the ultrastructure of cells. The chapter on the pituitary gland has been completely rewritten, to include the histochemical and experimental evidence for the differentiation of various cell types in the pars distalis together with a very brief account of modern concepts of neurosecretion and hypothalamo-hypophyseal relations.

Cowdry's textbook differs essentially from other textbooks of histology, in that it places more emphasis on histology as an auxiliary science to anatomy, physiology and pathology than on the study of morphological detail. The book is an admirable introduction to histology as a science, and there are some good chapters on "Methods of Studying Histology" and "Cells as Vital Units", but it should certainly not be used as a reference book. To mention a few examples, one would look in vain for a description of the structure of the carotid body or the wall of arterio-venous anastomoses or for an account of the cytology of the ovary. Its usefulness as a student textbook depends on the nature of the histology course given; but where, as in many university courses, the prevailing view is that a thorough knowledge of structure is a prerequisite for a proper understanding of function, this book cannot be recommended as a basic text-

book. However, an interested and intelligent student should benefit from reading this book in addition to one of the usual textbooks. A valuable feature of this book is the extensive list of references.

Biochemistry for Medical Students. By William Veale Thorpe, M.A. (Cantab.), Ph.D. (Lond.); seventh edition; 1960. London: J. & A. Churchill Ltd. 8″ × 5″, pp. 560, with 50 illustrations. Price: 30s. net (English).

THIS well-known book has been in use for over twenty years. The form and size in this edition remain unchanged. The chief alterations are in the sections on the interrelations between the principal metabolites, on oxidations and reductions in the body, and on hormones and vitamins.

The subject matter is considered in three main divisions: physical principles and the chemistry of the more important constituents of foods and tissues; digestion, absorption and intermediary metabolism (including respiration); nutrition, energy metabolism and excretion.

The information on the cost of foodstuffs and the relation of diet to income refers to conditions in Britain, and is not relevant in this country. The inclusion of the sometimes lavish American recommended dietary allowances (1948), as well as the more realistic figures of the Nutrition Committee of the British Medical Association (1950), seems unnecessary.

The author of a book such as this is subject to many unavoidable limitations. Thorpe's selection of material has proved to be well balanced. His revised list of 100 references to other textbooks and monographs has been classified so as to make its use easy by the student who wishes to extend his reading.

Progress in the Biological Sciences in Relation to Dermatology. Edited by Arthur Rook, M.D., M.R.C.P.; 1960. Cambridge: University Press. 9¼″ × 6″, pp. 496, with illustrations. Price: 84s. (English).

THIS book is a record of the papers delivered and the ensuing discussion at a course given on this theme in the Post-Graduate Medical School of the University of Cambridge from September 22 to 29, 1958. Subjects dealt with included: the melanocyte and melanogenesis; cutaneous innervation; the histochemical investigation of the skin; bacteriology and mycology; psychophysiological mechanisms; comparative medicine; immunology; inflammation; carcinogenesis; radiation and the skin; pharmacology. In each instance papers were read by men who are actively engaged in basic research on the subject, and the discussion of their ideas by clinicians makes reading of exciting interest.

In a paper on the structure of the skin in relation to its innervation, Graham Weddell points out that, though we are still quite ignorant as to the mechanism of paræsthesiæ or itch, it would be logical to regard them as resulting from disturbance in the regular pattern of activity reaching the brain rather than from the activation of a particular group of terminals and fibres (rather like radio static). The sensory nerves, like the skin itself, are labile and may adapt themselves, in both structure and function, to their surroundings.

Russell Davis, in discussing psychological mechanisms in psychosomatic disorders, stresses the frequent though not invariable stereotyping of reactions. He points out that, though it is the tradition in medicine to relate differences in disease to differences in pathogenic agent, attempts to explain psychosomatic reactions similarly have had limited success. Whitlock rather deprecates the acceptance of the importance of symbolism, pointing out that, whilst such interpretations are useful in psychotherapy, what is pragmatically useful is not necessarily scientifically true. He makes some interesting observations on the theory of central itching with feed-back to an area of skin already vulnerable because of previous injury or disease.

Four absorbing papers and discussions deal with carcinogenesis (of the skin in particular) and the fundamental effects of radiation on the skin. W. E. Parish indicates that autosensitization to skin has not yet been proved in the laboratory. Spector points out the importance of endogenous mechanisms in inflammation (recently emphasized in fungous infections by J. Walter Wilson), and Schacter ally active agents which can produce erythema, pain, œdema and pruritus. The section on comparative dermatology, in which are described an inherited disorder of the coat of guinea-pigs and "scrapie", a nervous disorder of sheep, describes the facts concerning some releasable pharmacologic- emphasizes the increasing interest in this subject, particularly in Cambridge.

This book is required and fascinating reading for all dermatologists. Parts of it can be read with the feet up.

Treatment of Cardiovascular Emergencies. By Aldo A. Luisada, M.D., and Leslie M. Rosa, M.D.; 1960. New York, Toronto and London: McGraw-Hill Book Company, Inc. 6¾″ × 3¾″, pp. 128. Price: not stated.

THIS work sets out to deal with the therapy of cardiovascular emergencies. Unfortunately, the authors have failed to present their material in a manner which will be useful to the people most likely to seek guidance from such a book—namely, the busy general practitioner and the hospital resident medical officer. The subject matter is confusing, and the authors' reasoning is often hard to follow. Furthermore, it is hard to agree with much of their recommended therapeutic regime. For example, they make the unqualified statement that in all cases atrial fibrillation without heart failure should be converted to sinus rhythm by the intravenous injection of quinidine. Even if it is assumed that the first half of the statement is correct—and it is surely against British cardiological teaching—the routine intravenous use of quinidine, as recommended by these authors, cannot be justified. It is also difficult to agree that quinidine should be given intravenously in supraventricular tachycardia. One cannot understand what the authors mean when they talk about Stokes-Adam attacks occurring without a change in pulse rate. The use of sitz baths for pulmonary œdema cannot have any place in modern medical practice. "Arfonad" is mentioned twice, without any reference to its dosage or method of administration. The reader struggling for guidance cannot be but confused when he reads in one chapter that nor-adrenaline is diluted for intravenous use in the ratio of 5 mg. to 500 ml., 8 mg. per 1000 ml. in another chapter, and 8 to 16 mg. in 500 to 1000 ml. in yet another. Electrical defibrillators and pacemakers are discussed, but the reader is not told how to perform cardiac massage; in an emergency, a scalpel is much more likely to be available. This book cannot be recommended.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Principles of Surgical Nursing", edited by Thomas F. Rose, M.S., F.R.C.S., F.R.C.S.E., F.R.A.C.S., F.A.C.S.; 1960. Sydney: Dymock's Book Arcade Ltd. 8¼″ × 5¼″, pp. 642, with many illustrations. Price: 45s.

"Factors Controlling Erythropoiesis", by James W. Linman, M.D., and Frank H. Bethell, M.D.; 1960. Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications Ltd. 9″ × 6″, pp. 218, with many illustrations. Price: 66s.

"Nucleoproteins", Proceedings of the 11th Solvay Conference on Chemistry, Brussels, June 1-6, 1959. New York: Interscience Publishers Inc. 9¼″ × 6¼″, pp. 364, with many illustrations. Price: $10.50.

"Bedside Medicine", by I. Snapper, M.D.; 1960. New York: Grune and Stratton, Inc. 9″ × 6¼″, pp. 606. Price: $14.50.

"The Central Nervous System and Behaviour: Transactions of the Third Conference February 21, 22, 23 and 24, 1960", edited by Mary A. B. Brazier; sponsored by the Josiah Macy, Jr. Foundation and The National Science Foundation; 1960. 9″ × 6″, pp. 476, with many illustrations. Price: $7.50.

"Leukemia Cutis", by Samuel M. Bluefarb, B.S., M.D., F.A.C.P.; 1960. Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications Ltd. 8¼″ × 5¼″, pp. 516, with many illustrations. Price: £7 8s.

"Auditory Disorders in Children: A Manual for Differential Diagnosis", by Helmer R. Myklebust; third edition; 1960. 8″ × 5″, pp. 380. Price: $7.75.

"The Queen Charlotte's Textbook of Obstetrics", by Members of the Clinical Staff of The Queen Charlotte's Maternity Hospital, London; tenth edition; 1960. London: J. & A. Churchill Ltd. 9¼″ × 6″, pp. 544, with 240 illustrations. Price: 50s.

"Children for the Childless: A Concise Explanation of the Medical, Scientific, and Legal Facts about Conception, Fertility, Sterility, Heredity, and Adoption", edited by Morris Fishbein, M.D.; 1960. London, Melbourne and Sydney: William Heinemann Ltd. 8¼″ × 5¼″, pp. 224. Price: 15s. 6d.

The Medical Journal of Australia

SATURDAY, FEBRUARY 11, 1961.

BREAKING THE NEWS.

SIR FREDERICK TREVES[1] tells the story of a pretty young woman who burst unannounced into his consulting room, where he was interviewing a patient. She was carrying a little girl under her arm and without preliminaries exclaimed: "He wants to take her foot off." When the situation was sorted out, it appeared that the pretty child of the pretty mother had tuberculous disease in one foot. A young surgeon who had been consulted appeared to have let fall some rash remark about taking the foot off. The mother reacted violently. As Treves puts it: "She, whom I came later to know as one of the sweetest and gentlest of women, changed at the moment to a wild animal—a tigress." Snatching up her baby girl without more ado she rushed off to Treves, whose name she had been given as a possible person to consult. So worked up was she that she flung herself into his room, quite unconscious of anything—even of herself or of her considerable good looks. Treves continues:

> The mother's fury against my surgical colleague was almost inexpressible. If the poor man had suggested cutting off the child's head he could not have done worse. "How dare he!" she gasped. "How dare he talk of cutting off her foot! If he had proposed to cut off my foot I should not have minded. It would be nothing. But to cut off her little foot, this beautiful little foot, is a horror beyond words, and then look at the child, how sweet and wonderful she is! What wickedness!" It was a marvellous display of one of the primitive emotions of mankind, a picture, in human guise, of a tigress defending a cub. By a happy good fortune, after many months and after not a few minor operations, the foot got so well that the glare in the eyes of the tigress died away and she remembered again that she was a pretty woman.

This is perhaps not a usual pattern of the reaction of a person faced with bad news from a doctor, but it emphasizes the depth of the reaction that is likely to take place. In some cases hardly a ripple is seen on the surface, in others there is a complete boil-over. It depends on the individual (patient or patient's relative or friend) and on the doctor who breaks the news. The individual may be prepared and later helped by an understanding doctor, but basically that side of the reaction is outside the doctor's control. Much, however, remains in the doctor's hands. Maurice Davidson, in an essay on "What to Tell the Gravely Ill Patient",[2] has written: "That which hurts most, in the delivery of bad news, is not what the doctor says but the manner in which he says it." This would appear to have been the essence of the matter in Sir Frederick Treves's story and is emphasized by Sir William Upjohn in a short but valuable paper published in this issue (see page 195). What is told is important, but it is the lesser factor, at least at the time of telling. The apprehensive patient may be upset by anything; the well-balanced, courageous or phlegmatic patient may not even blink at the worst news. The clumsy doctor can make almost any diagnosis or prognosis frightening; the understanding doctor can soften the hardest blow.

At the same time much thought may need to be given to how much the patient should be told. If major surgery, especially of a mutilating character, is to be recommended, the patient will almost always need to know the diagnosis and to accept in advance, so far as he is able, the implications of the proposed operation. It is a shocking thing, as Sir William Upjohn says, for a patient to awake from an extensive operation without having been warned beforehand of the possibilities. Unpleasant though the task may be, it cannot be shirked, whether the responsibility is accepted by surgeon or by family doctor. A patient who is going to die soon is also surely entitled to know if he wishes to. It may not be easy for the doctor to know the patient's wishes here, and such information should not be forced on a patient, but the doctor must be prepared to accept the challenge when it comes. It is a widespread experience that many patients quietly deduce for themselves the fact that they have an incurable disease or one with at least an ominous prognosis; they may not want to talk about it or even to admit it too clearly to themselves, but their questions are entitled to be answered when they come, albeit gently and with restraint. The situation in which relatives forbid a doctor to tell a patient he is likely to die is a difficult one, and what the doctor does will depend on the particular circumstances, but he has no right just on his own account to withhold bad news. There are many good reasons why a person may want to know and need to know that death is imminent, and it is important not to overestimate the apprehension by any individual of this universal phenomenon. It is another matter, of course, where there is doubt about a diagnosis, and any ray of hope should be fostered. Certainly the fears of a patient who may have made a wrong and gloomy surmise about his condition should be allayed. But whatever is said or done, crudeness or disregard of the feelings of either the patient or those who care about him is unforgivable and professionally damnable.

It is important not to confuse the question of the patient who is almost certainly going to die with that of the patient newly diagnosed as suffering from such a condition as cancer. Indeed, the patient's tendency to think of a

[1] "Breaking the News" in "The Elephant Man and Other Reminiscences", Cassell, London, 1923.

[2] In "Medical Ethics", edited by M. Davidson, Lloyd-Luke, London, 1957: 112.

diagnosis of cancer as a death sentence should be anticipated, and, so far as is right, forestalled. Nevertheless, the gravity in a patient's mind of a future shadowed by cancer will always be considerable, and a doctor cannot think too much about the handling of such situations. Fuller discussions on the subject, in addition to Sir William Upjohn's paper, are to be found in the essay by Maurice Davidson already mentioned and others in the same book, in a thoughtful discussion of "The Christian Approach to the Disabled, the Incurable, and the Dying", by Anthony P. Waterson,[3] and in the report of a Mayo Clinic Symposium[4] including the views of an internist, a surgeon, a psychiatrist, a pædiatrician and others. Sound advice, including a warning against giving definite prognosis, also appears in a paper by John Hayward,[5] delivered at the Hobart Congress in 1958. It will suffice for now to sum up the situation by quoting a statement of the internist, Edward H. Rynerson, contributing to the Mayo Clinic symposium and emphasizing that every case has to be dealt with on its merits: "There can be no rule. There can only be a physician with a heart—a physician who, as a person, wants to be of service to another human being."

Current Comment.

MIGRANT CHILDREN IN AUSTRALIA.

THERE is much of interest to be found in the first report on the progress and assimilation of migrant children in Australia,[1] an interim report which was presented to the Australian Citizenship Convention at Canberra in February, 1960, by a committee apparently set up by the Department of Immigration. The membership of the committee is: The Hon. Mr. Justice Dovey (Chairman), Sir Richard Boyer, K.B.E. (Good Neighbour Council of Australia), Hon. P. J. Clarey, M.P., Dr. J. R. Darling, C.M.G., O.B.E., A. J. Lee, Esq., C.B.E., M.C. (National Executive, R.S.S. & A.I.L.A.), A. E. Monk, Esq. (President of the A.C.T.U.), and Mrs. J. G. Norris, O.B.E. (National Council of Women). The terms of reference of the committee were:

(*a*) To investigate and report upon the progress and assimilation of migrant children and the Australian-born children of migrant parents, taking into consideration such aspects as—

(i) their educational and general progress in Australian schools; cultural, sporting and social activities and general participation in the life of the Australian community;

(ii) any special problems which may be foreseen, e.g., difficulties arising in the home life of migrant families as a consequence of resettlement;

(iii) the standard of behaviour generally and the incidence of delinquency amongst migrant children.

(*b*) to suggest any measures which might be taken to facilitate satisfactory assimilation.

The committee collected its information in a variety of ways. A large part of the report is based upon interviews by members of the committee with a wide range of people from a variety of spheres, including educational authorities, police, child welfare and health authorities, various trade unions, and sporting and social bodies. To supplement this information, in areas where apparently inadequate data had been collected from interviewers, the committee undertook a number of surveys itself. These were concerned with the assimilation of pre-school children and the incidence of delinquency among migrants. The result is the present document, which the committee designates an interim report; because of the magnitude of the task and its continuing nature they hope to maintain their study, and presumably they will be issuing further reports.

Some of the findings make interesting reading. It is reported that about 97% of young migrants settle down well in Australia, that their scholarship is above that of the average Australian-born child, and that delinquency if anything is less of a problem than among young Australians. On the debit side the committee found that in social activities both migrant children and their parents lag far behind Australians, and that one of the barriers to rapid assimilation is the fact that in a great many homes English is not spoken, so that the children have little opportunity to practise the English that they learn at school. This also must have an effect on the assimilation of adults into the community, and in particular their participation in activities that are associated with the life of their children. It is well recognized that the active association of parents in school functions is a source of pride and satisfaction to most children, raising their status in the eyes of their peers.

There are, however, some disturbing factors about this type of investigation. Members of the committee are extremely able and well-known citizens, but most are without any special training in the social sciences, and it must surely be admitted that the problem of the assimilation of migrants, whether adults or children, is largely in the field of the social sciences. In this age, which prides itself on being highly scientific, it is unreasonable to expect scientific accuracy in the collection of data relating to physical, chemical and biological studies and not to expect the same approach to problems in the field of the social sciences. More particularly is this disturbing when it is realized that there are, in most of the Australian universities, a number of departments which could provide teams of highly qualified people to make accurate observations and collect data in such a way that it could be replicated in a number of places or at different times in the future.

This report should be accepted for what it is—an interim report, which has stated a number of problems. Further investigation of these, however, should be entrusted by the committee to qualified scientists, rather than that the investigation be carried out by members of the committee. We also note that although reference is made in the report to the significance of health and the use made by the parents of health centres, a surprising omission from this report, from the viewpoint of readers of this journal at least, is the absence of any reference to the standard of health, either physical or emotional, of migrant children.

TREATMENT OF CARCINOMA OF THE BLADDER.

THE treatment of carcinoma of the bladder is a matter which has exercised the ingenuity and resourcefulness of urologists for many years, and is likely to do so for many years to come. More so, perhaps, than with most cancers, the treatment of this condition has to be varied according to the circumstances of the individual case. Many lines of treatment are available, but none are appropriate to more than a limited selection of cases. In a paper read before the Northern Section of the American Urological Association, Sir Eric Riches,[1] of the Middlesex Hospital,

[3] In "Ideals in Medicine", edited by V. Edmunds and C. G. Scorer, Tyndale Press, London, 1958: 90.

[4] *Proc. Mayo Clin.*, 1960, 35: 239 (May 11).

[5] MED. J. AUST., 1959, 1: 14 (January 3).

[1] "First Report on the Progress and Assimilation of Migrant Children in Australia", by a Special Committee of the Commonwealth Immigration Advisory Council; 1960. Canberra: The Commonwealth Immigration Advisory Council. 10¾" × 8¼", pp. 44.

[1] *J. Urol.* (*Baltimore*), 1960, 84: 472 (September).

London, has presented a review of the choice of treatment in carcinoma of the bladder. In this, he covers the whole range of treatment applicable to various degrees of severity of vesical carcinoma, and relates the choice of treatment to the results. At the outset he emphasizes that good results are an indication that the right type of treatment was chosen for that particular growth. He states that before treatment is started, it is vital to impress on the patient the need for regular follow-up for the rest of his life. He considers that the patient is more likely to observe this if it is hinted to him that the growth has "some malignant characteristics". While a cure is aimed at, often one has to be content with a reasonable measure of control or palliation. A simple method should be chosen rather than an elaborate one, if it is likely to be effective, and the patient should, if possible, be left with a functioning bladder.

For papillary tumours of low-grade malignancy and moderate size, especially if pedunculated, endoscopic removal or destruction is recommended. Riches' results by this method in 153 cases show an over-all five-year survival rate of 60%. For a higher histological grade of malignancy, as observed by biopsy, open approach, with interstitial irradiation by radon seeds, is necessary. The result in 100 patients treated this way shows an over-all survival of 59%. It is evident from this that, for over-all survival, there is little to chose between the two methods just described, and this applies to tumours of low malignancy. However, in those of higher malignancy, the interstitial application of radon gave better results than endoscopic treatment. Thus, 75% of patients treated endoscopically needed further treatment, and only 42% of those treated by radon did so. At the same time, it must be remembered that neither of these methods necessarily prevents distant metastases.

More radical open methods are often indicated, but, with papillary tumours, the greatest care must be taken to avoid spilling tumour cells into the wound. Open localized excision is useful when the growth is too extensive for endoscopic diathermy, or the patient is unfit for cystectomy. In this group 30% survived five years. Partial cystectomy—that is, removal of the whole thickness of the bladder wall around and clear of the growth—is useful in large papillary growths. Even if the lower end of a ureter has to be excised and the duct reimplanted, Riches advised that this should be done. As for total cystectomy, this is essential if one wants complete cure of multiple and extensive papillomata. However, it carries with it the need for urinary deviation and its added risks. In this particular papillary group there were 38 cases, and there is a five-year survival rate of 42%. Riches notes that despite the reluctance of many patients to have an external urinary fistula, most of them manage it extremely well and are able to follow their normal occupations. It should be noted that recurrence of papillary tumours in the bladder may be due to a small unsuspected papillary tumour of the renal pelvis. Intracavitary irradiation from a central source of cobalt-60 placed in a balloon has proved disappointing. As for external irradiation by supervoltage therapy, out of 25 cases, in 14 of which the tumours were purely papillary and the rest papillary and solid, good palliation was obtained in 13 and poor palliation in 12.

Solid tumours have a higher intrinsic malignancy than papillary; they are curable only if seen early, before invasion of surrounding tissues, blood vessels and lymphatics has occurred. Riches states that they are not amenable to endoscopic methods or to interstitial irradiation with radon. Implantation of radium needles within the bladder is of no use because of the severe vesical reaction, but there is a place for extravesical implantation of radium needles. This can render the organ more suitable for total cystectomy after some months. If the site of the tumour is suitable, partial cystectomy may be indicated and useful; it was used in 88 cases in 64% of which muscular invasion had occurred. The five-year survival rate was 36%. With perivesical spread there were no five-year survivors. Where there is deep infiltration of the bladder wall, total cystectomy is no more curative than partial, and the results are poor for total cystectomy in this group. Riches states that he has done 120 total cystectomies; the five-year survival rate depends almost entirely on the depth of penetration of the tumour of the bladder wall. In the solid infiltrating growth there is only a 9% five-year survival rate, but there is quite a degree of palliation.

In discussing external irradiation, Riches refers to patients given supervoltage therapy in collaboration with Professor Brian Windeyer; during the four years to the end of 1958, 43 patients were so treated. The tumours were of the following varieties: 14 papillary, 25 solid, 11 mixed papillary and solid, 3 purely squamous. Three-quarters of them had solid elements, which shows that the worst cases were being treated. The aim of treatment was to give about 7000*r* over about two months. There was good palliation for rather less than one half of the patients, but the list of distressing complications was rather formidable, and Riches appears to consider the results discouraging. He states that since these results began to come through, he is again doing more total cystectomies.

In his concluding remarks Riches states:

> I still feel that the ultimate treatment of this serious disease is going to lie in a combination of radiotherapy and surgery. The two types, papillary and solid, differ so much in their behaviour as to form two distinct diseases, the one curable or controllable by many different methods, the other having such intrinsic malignancy as to defy treatment unless seen very early. Although it might appear that the results depend more on the malignancy of the growth than on the nature of the treatment it should be possible to control either form if it is diagnosed early, fully assessed, boldly treated and followed up with determination.

STUDY GRANTS FOR AUSTRALIAN GENERAL PRACTITIONERS.

AN unusual but worthwhile innovation in the matter of study grants has been announced by the Post-Graduate Medical Foundation in the University of Sydney. It has accepted an offer by one of its Governors, Mr. R. F. Peters, on behalf of The Upjohn Company (Australia) Pty. Ltd., to sponsor each year a number of grants for general practitioners. The purpose of these is to assist general practitioners throughout Australia in furthering their post-graduate medical study by enabling them to attend specified post-graduate courses. They will be known as the Upjohn Grants for General Practitioners' Post-Graduate Study.

For 1961 ten grants of £100 each will be made available and will be distributed approximately in the following proportions: New South Wales three, Victoria three, South Australia, Queensland, Western Australia, Tasmania one each. The specified course for 1961 will be the General Revision Course to be held in Sydney from May 8 to 19.

In future years the Upjohn Company proposes to increase its contribution so as to extend the number of grants, to assist more general practitioners and to expand the scheme to cover attendance at courses in other States. For 1961 the grants will be administered on behalf of the Foundation by the Post-Graduate Committee in Medicine in the University of Sydney, in association with the Australian Post-Graduate Federation in Medicine, which represents all the Post-Graduate Committees of Australia. Conditions of the grants and application forms may be obtained from the Director, The Post-Graduate Medical Foundation, Herford House, 188 Oxford Street, Paddington, to whom all applications should be forwarded by March 3, 1961. These grants, the first of their kind made for general practitioners, are, as Dr. V. M. Coppleson, the President of the Australian Post-Graduate Medical Foundation, has pointed out, an outstanding contribution to the advancement of post-graduate medical education in Australia.

Abstracts from Medical Literature.

MEDICINE.

Polyvalent Antigen against Staphylococcal Infections.

L. GREENBERG AND M. Y. COOPER (*Canad. med. Ass. J.*, July 23, 1960) state that attempts to produce an effective prophylactic against staphylococcal infections have been in progress for almost 80 years, but that none has proved particularly successful. The approach to the problem has been invariably based on procedures which have been found successful against other bacterial diseases, and has been hampered by lack of knowledge concerning the fundamentals of the pathogenesis of staphylococcal disease. Vaccines consisting of killed suspensions of whole bacteria, antigenic fractions of *Staphylococcus aureus* and staphylococcal toxoids have been prepared, but their continued use has been an indication of the failure of other therapeutic measures and not of success of active immunization. The authors have developed a polyvalent somatic (intracellular) antigen by combining the enzyme-lysed fractions of a number of vaccines prepared from different phage types of *Staph. aureus*. They describe the procedures used and report the results obtained in animal experiments. They state that this antigen will protect experimental animals against challenge with both lethal and skin-infecting doses of 36 test cultures, representing all the important human phage types of *Staph. aureus*. The results in animals have been so encouraging that preliminary trials in human subjects have been started. The authors state that their studies show that antibodies to intracellular antigens play a greater part than antibodies to either the cellular or the extracellular antigens in the prevention of staphylococcal disease, and that in every instance somatic antigen staphylococcal vaccines gave better protection than their homologous bacterial vaccines. Toxins and toxoids played no part in the somatic antigen vaccine used. Polysterene latex antigens were prepared from five separate strains of different phage types of *Staph. aureus*, and agglutination tests indicated that all of the strains have a common agglutinating antigen.

Effects of Resection on the Hypertension in Coarctation of the Aorta.

H. W. MARCH *et alii* (*Brit. Heart J.*, June, 1960) report a study on the blood pressure response of successful resection in cases of coarctation of the aorta. The authors state that there is a slow gradual fall in the blood pressure which may continue for several weeks, and that at the time of leaving hospital only one-third of patients have a normal blood pressure. They found that the post-operative course was not infrequently interrupted by paroxysmal attacks of hypertension and the development of severe abdominal pain. The latter may occasionally go on to arteriolar vascular necrotic changes similar to polyarteritis nodosa, possibly a stretch effect due to increased intravascular tension. Two patients died within six weeks of operation with severe abdominal hæmorrhage. The authors discuss the mechanisms of coarctation hypertension and suggest that the observed behaviour can be explained by hæmodynamic factors caused by the coarctation itself, without invoking renal ischæmia or increases in peripheral vascular resistance.

The Opening Snap in Mitral Incompetence.

P. G. F. NIXON *et alii* (*Brit. Heart J.*, June, 1960) describe 12 patients in whom mitral regurgitation was caused by inability of a pliant, mobile aortic cusp to meet a shrunken and immobile mural cusp, a variety of mitral regurgitation amenable to modern surgical treatment. Each patient presented clinically with a loud opening snap and pan-systolic murmur of mitral regurgitation. If further experience proves this association of signs reliable in the diagnosis of incompetence caused by the disease of the mural cusp, it may well be of value in the selection of patients for operation. The opening snap indicates pliancy and mobility of the aortic leaflet. The opening snap is not diagnostic of either pure stenosis or of commissures that split readily. Whether or not the mural cusp plays an effective part in closing the normal mitral orifice, considerable regurgitation may be caused by its shrinkage from rheumatic heart disease.

Patent Ductus Arteriosus in an Octogenarian.

J. BØE AND S. HUMERFELT (*Acta med. scand.*, May 20, 1960) report the case of a woman aged 80 years in whom a diagnosis of patent ductus arteriosus was made more than 50 years ago, when she complained of fatigue, dyspnœa and palpitation on exertion. Evidence of heart failure was present in her younger years, but apart from a few admissions to hospital, she managed to lead an active and hard-working life as a nurse up to the age of 68 years. At the age of 70 years she was admitted to hospital with acute pneumonia, and on this occasion the diagnosis was confirmed by cardiac catheterization. The authors state that at the time of writing the patient had recently shown signs of increasing cerebral arteriosclerosis, but that she was still able to keep house for her three nephews.

Diabetes Mellitus.

F. S. PERKIN (*J. Amer. med. Ass.*, May 7, 1960) discusses the practical use of hypoglycæmic agents. Diet suitable to the patient's height, weight, sex and physical activity is the most important factor in the treatment of diabetes. Regular insulin is necessary in treating acidosis and coma; the lente insulins and other derived insulins may be used also. The group of sulphonylureas which includes carbutamide and tolbutamide must be used with caution as there is evidence that they may not be the best treatment for individual patients. Insulin must be used with care, and over-dosage must be avoided as well as under-dosage. Ordinary or regular insulin may be used in proper combination with protamine zinc insulin, and lente insulins have their place. It is unwise to use more than 60 to 70 units of any insulin in one dose in regular treatment. In juveniles, two doses of insulin daily, two-thirds before breakfast and one-third before the evening meal, are usually best. Of the orally given sulphonylureas, the use of carbutamide has now been discontinued; tolbutamide is still the most widely used; metahexamide was abandoned on account of late hepatic effects. There are many qualifications as to the type of case in which sulphonylureas are the drugs of choice. Tolbutamide is said by some authors to be effective in early and mild diabetes of juveniles. The general opinion is that tolbutamide is most reliable in non-ketotic asymptomatic diabetics of nearly normal weight, over 40 years of age and taking less than 40 units of insulin daily. Chlorpropamide ("Diabinese") is powerful, but has not been long in use. Phenformin (*n*-phenethyldiguanide) is a new orally administered hypoglycæmic agent which has reduced insulin requirements. It may produce severe gastro-intestinal symptoms in 7% of patients or more. The author emphasizes the necessity for careful study of each individual patient.

Serum Cholesterol Levels in Human Atherosclerosis.

J. C. PATERSON, L. DYER AND E. C. ARMSTRONG (*Canad. med. Ass. J.*, January 2, 1960) report on the study of serum cholesterol levels at least once a year on 100 ambulatory patients between 1953 and 1959. There were 191 fatalities during this period, and in each of these the severity of atherosclerosis was determined at autopsy in four different arteries and with the use of six different criteria. The *ante mortem* serum cholesterol levels were then compared with the severity of the disease in the arteries, and with the presence or absence o those complications of atherosclerosis that could be detected at autopsy. The result lent little support to the contention that the severity of atherosclerosis is related to the level of serum cholesterol except perhaps when it exceeds 300 mg. per 100 ml. In 58 cases in the age group 60 to 69 years the relationship between the level of serum cholesterol and the severity of the disease was found to be significant only once in 40 statistical analyses, and the complications of atherosclerosis were just as frequent in patients with serum cholesterol levels of 150 to 199 mg. per 100 ml. as in patients with moderately high ones of 250 to 299 mg. per 100 ml.

The Electrocardiogram and Its Interpretation.

H. N. SEGALL (*Canad. med. Ass. J.*, January 2, 1960) has analysed the reports by 20 physicians on a set of 100 electrocardiograms. All the interpreters had had special experience in electrocardiography. They were asked to place the unknown tracings in one of the following three groups: myocardial infarction, within the normal range, or showing non-specific electrocardiographic abnormalities. There was 100% agreement in the reporting of 21 electrocardiograms, 90–95% agreement in 23 and 70–85% agreement in 33. As a result of this

study the author concludes that the ideal circumstances for interpreting are when the physician, after examining the patient clinically, records the electrocardiogram himself and then interprets it in the light of his full knowledge of his patient. Comparison with previous tracings is extremely helpful. In contrast, the conditions under which electrocardiograms are reported on in hospitals are far from ideal and are largely responsible for the wide range of differences of opinion.

Recurrent Infarction of the Heart.

G. BLOMQVIST *et alii* (*Acta med. scand.*, August 10, 1960) have followed the subsequent history of 970 patients with infarction of the heart over a total of 3278 observation years, during which there were 162 episodes of recurrent infarction. They found that the risk of experiencing a new attack was significantly higher during the first year after primary infarction than later. In men below 60 years of age the higher risk was mainly confined to the initial six months, whereas in the whole series there was practically no difference between the first and second half-year periods. The mortality in the four weeks after recurrent infarction did not differ significantly from that after primary infarction.

Myocardial Infarction during Treatment of Depression with Imipramine.

L. SLOMAN (*Canad. med. Ass. J.*, January 2, 1960) reports a patient who developed myocardial infarction during imipramine ("Tofranil") treatment for recurrent depression. Reviewing the literature, the author quotes eight other cases of cardio-vascular complications during imipramine treatment. However, reviewing the evidence he concludes that myocardial infarction may be coincidental in these cases, or possibly precipitated by other factors such as concurrently administered insulin sub-coma treatment.

Tuberculosis Epidemiology in Adolescence.

P. FRÉOUR *et alii* (*Rev. Tuberc. (Paris)*, May–June, 1960) have investigated the incidence of tuberculous morbidity amongst large groups of young men and women whose tuberculin reaction had been tested in high-school days. Contrary to the experience recorded by many authorities since the beginning of the century, they found that the incidence of disease was greater among the reactors to tuberculin than among the non-reactors. Persons whose reaction was due to vaccination with the B.C.G. were excluded from consideration. The authors attribute this finding to changed epidemiological climate, the population under review having a small incidence of tuberculosis, in contradistinction to the heavily tuberculized populations in which the same question has been investigated in the past.

Guillain-Barré Syndrome.

W. PARKER *et alii* (*Canad. med. Ass. J.*, April 18, 1960) report the isolation of a Type 6 ECHO virus from the cerebrospinal fluid and from the fæces of a boy suffering from the Guillain-Barré syndrome. Supporting evidence that the virus was the cause of the disease (in the form of a significant rise in the titre of antibodies against this virus during the course of the illness) was lacking, though this may have been due to steroid therapy. The authors consider that the syndrome may be caused by any one of a variety of viruses which normally are not primarily pathogenic to the cells of the central nervous system and therefore do not produce irreversible damage.

Congestion of the Female Urethra in Heart Failure.

R. A. WENTZELL (*Canad. med. Ass. J.*, September 3, 1960) draws attention to a rather common but infrequently recognized syndrome, seen in elderly women with congestive heart failure. Œdema of the urethra and of the tissues about the urethra and bladder neck causes complaints of urgency, frequency and mild dysuria, and sometimes of stress incontinence or of suprapubic discomfort. A few leucocytes may be found in the urine, but culture is usually sterile. The author states that it often happens that the patient is treated energetically for bacterial cystitis without avail while treatment for the cardiac failure is not energetic enough.

Tuberculosis in Singapore.

COTTER HARVEY *et alii* (*Brit. J. Dis. Chest*, July, 1960) report a pilot survey conducted in Singapore to estimate the incidence of tuberculosis on the island. A total of 54,812 persons was X-rayed, of whom 2058 or 3·75% were found to be suffering from active tuberculosis. In addition 2064 cases of inactive tuberculosis and 323 cases of heart disease were found.

Long-Term Anticoagulant Treatment.

R. L. MCMILLAN *et alii* (*Canad. med. Ass. J.*, September 10, 1960) report the preliminary results (after two years) of a controlled study to determine the value of prolonged anticoagulant treatment after infarction of the heart. The authors state that to date the treatment has failed to prevent further episodes of infarction or to save life. Hæmorrhage was a serious and not infrequent complication of the treatment, and it is pointed out that any benefit of treatment must be balanced against the danger of bleeding. The authors comment that these results are at variance with the more favourable experience reported in larger controlled studies, and discuss certain inconsistencies in these series.

Psycho-Physiological Mechanisms in Hypertensive Vascular Disease.

A. P. SHAPIRO (*Ann. intern. Med.*, July, 1960) discusses the psychophysiological mechanisms in hypertensive vascular disease. It appears that there is ample evidence that physiological pathways exist by which emotions can be translated into blood pressure elevation, and that such effects are responsible for many of the acute and even chronic rises in blood pressure seen in hypertensive individuals, and probably aggravate the disease. It seems unlikely that psychogenic disturbances are ever solely responsible for the original development of hypertensive vascular disease, but the evidence suggests that these influences act as contributing causes. Supportive psychotherapy has something to offer in the management of hypertensive disease, including amelioration of symptoms, slowing of progression, avoidance of iatrogenic noxious stimuli, and adjustment of the patient's attitude to his environment and his disease. Knowledge of the impact of emotional variables should serve to illustrate to the physician that he does have some control of the situation and should help him to manipulate the doctor-patient relationship as circumstances dictate. In certain instances, most typically in the young individual with labile levels of blood pressure, more intense psychotherapy is warranted. This does not obviate the necessity of the intelligent use of hypotensive medications, but rather it may simplify the physician's task in the employment of these drugs. The author points out that the psychophysiological mechanisms involved in hypertensive disease are not magical manifestations, but are subject to investigation by the same scientific principles employed in other disciplines, and urges that when this is done properly, the observations should command the respect and attention of all clinicians and investigators — both the "psychiatrically oriented" and the "organically minded" —who are interested in advancing the basic understanding of hypertensive vascular disease.

A New Face of Tuberculosis.

F. T. ROQUE (*Amer. J. med. Sci.*, July, 1960) discusses a new face of tuberculosis. The clinical notes of four studied cases are presented. As a result of the use of high doses of isoniazid (10 to 16 mg. per kg. of body weight) in combination with daily PAS or streptomycin, or both, in the treatment of pulmonary tuberculosis, complete clearing of the disease in about 30 days may occur. A note of warning is issued as to the danger of changing the diagnosis in such cases, especially in those in which the ætiological organisms were not obtained.

Acute Infectious Arthritis in the Aged and Chronically Ill.

R. F. WILLKENS *et alii* (*Arch. intern. Med.*, September, 1960) discuss acute infectious arthritis in the aged and chronically ill and present the case histories of six patients from among a series of 19 patients treated in the last five years. This disease is not rare in elderly, debilitated patients, and usually develops by hæmatogenous spread from other foci of infection. A high index of suspicion and joint paracentesis with adequate synovial fluid examination constitute the essential diagnostic elements. Adequate systemically administered antibiotics gave good results in patients infected with pneumococci, but no single regime was found uniformly successful in *Staphylococcus aureus* joint infections. However, vancomycin is thought to warrant further clinical evaluation in the latter cases.

Brush Up Your Medicine.

INFECTIOUS HEPATITIS.

THE introductory paragraph to an article on this subject, written in precisely the same circumstances in the Journal of February 18, 1956, read as follows:

> The present epidemic of infectious hepatitis is rightly receiving considerable attention because the more attention that can be given to a disease spread by dirtiness, the more chance there is of preventing future epidemics. Outbreaks of hepatitis have been common for centuries—one was described twelve hundred years ago by the Pope, and this ancient history should make us pause to consider how little progress we have made in preventing epidemics of communicable diseases in peacetime, especially those whose transmission is related to poor personal and public hygiene.

There is no reason to alter this in any way; indeed, the present epidemic serves to emphasize what was written four years ago.

Epidemiology.

Acute infectious hepatitis appears to be caused by a specific virus. It occurs sporadically and in epidemics with an incubation period of two to six weeks, in contrast to the closely related serum hepatitis which has an incubation period of two to six months.

The virus has been isolated from the serum and fæces of patients in the acute phase of the disease and has been demonstrated to be present in convalescent serum for months. It is important to remember that patients may be infectious before, during and for a long time after an acute attack. Symptomless carriers, particularly school children and infants, are not rare and are most important disseminators of the disease.

The route of infection is usually intestinal; hands, water, milk and food contaminated by fæces are the common vehicles of transmission. Carriers and symptomless patients are most important as they are not usually detected. The spread is from person to person, particularly by contaminated hands.

Lasting immunity follows an attack, but there appears to be no cross-immunity between acute infectious hepatitis and serum hepatitis.

Epidemics are especially prone to occur in institutions, families, and communities where people are living in close contact, poor hygiene playing a particularly important role.

Some Clinical Features.

Whilst children and young adults appear to be most often affected, no age group is immune. Typically the disease has a preicteric stage of anorexia, fatigue, distaste for tobacco and alcohol, abdominal discomfort or pain and malaise, and a succeeding icteric stage with symptomatic improvement. Fever and chills are common in the earlier stages and jaundice may be the presenting feature, but it may be absent. Aches and pains in the limbs, transient lymphadenopathy, some rashes and severe headache may all occur and may be quite confusing. Itching is uncommon, but tends to occur with the onset of jaundice and usually lasts only a few days. Bleeding phenomena may be severe and may confuse the diagnosis. The early gastro-intestinal symptoms may closely mimic a variety of other disorders and not suggest the real diagnosis.

In the average case, dark urine is an early sign, and careful testing reveals the presence of bilirubin, often before jaundice can be detected. Urobilinogen appears in the urine in increased amounts quite early. In the jaundiced patient the stools tend to be pale, and a return of colour is often a sign of recovery.

Apart from jaundice and bile in the urine, the most important physical finding is tenderness of the liver, which is best detected by pain on jarring the right lower part of the chest wall.

Biochemical tests of the blood may be of considerable value in the doubtful case, and they do help in the assessment of the changes taking place in the patient having a prolonged illness. The results of flocculation tests may take many weeks or months to return completely to normal, abnormal results persisting long after the clinical evidence of activity has disappeared; these circumstances do not occasion alarm, but rather suggest continued follow-up. If one suspects continued activity, liver biopsy should be carried out, because it is the only certain way of discovering exactly what is happening in the liver; but it is improbable that a patient with persistently negative biochemical findings will develop progressive hepatitis and cirrhosis.

Acute non-icteric hepatitis is common and presents as a gastro-intestinal disorder, especially in children. The presence of liver tenderness, of an epidemic, of a history of significant contact and of positive results from liver function tests assist in making the diagnosis but since all these phenomena may be absent, except for tenderness over the liver, the chief means of making a diagnosis is by clinical awareness. These patients are just as likely to become carriers as are icteric patients.

Hepatitis during pregnancy does not result in hepatitis in the child; indeed, maternal infectious hepatitis has little effect at all upon the child. On the other hand, the mortality of pregnant women with acute infectious hepatitis is very much higher than that of women as a whole.

Diagnosis.

In an epidemic the diagnosis is usually easy, and the errors are likely to be on the side of considering a patient with stone, carcinoma of the pancreas or chlorpromazine jaundice to have infective hepatitis. Diagnosis is primarily clinical and should be suspected in the pre-icteric stage, when tenderness over the liver and dark urine caused by slight bilirubinuria occur in a patient with malaise and gastro-intestinal symptoms. The absence of any history of contact in no way lessens the probability of infectious hepatitis, because many carriers are symptomless.

The value of the liver function tests is undisputed and they usually indicate a type of liver disease, but not its ætiology. They are especially useful to determine the progress and activity of the disease; but the average patient can be managed clinically if attention is paid to the liver, the jaundice, the symptoms and the urine.

A raised serum bilirubin level and the demonstration of bile in the urine are the most useful liver function tests for the diagnosis of acute infectious hepatitis. Very high levels of serum transaminase activity are usual and are of considerable diagnostic value, because a raised level is taken to indicate the presence of active parenchymal cell damage. The levels commonly fall rapidly to normal while other tests continue to show abnormalities. The flocculation tests are useful and usually give positive results, and the bromosulphthalein retention is increased. It must be very rare indeed for all liver function tests to give negative results during the acute phase of the disease in adults, although standard tests are reported to do so in infancy.

Infectious mononucleosis may be difficult to exclude, because there may be jaundice in this malady, and both cervical lymphadenopathy and the presence of abnormal lymphocytes occasionally occur in cases of acute infectious hepatitis. The course of the disease, the degree of jaundice, the extent of lymphadenopathy, the Bunnell-Paul test and liver function tests help to differentiate in the difficult case.

Anicteric patients present major diagnostic problems, and in the absence of positive results from liver function tests, a liver biopsy may be the only way to make a positive diagnosis. However, this can hardly be recommended as a routine. The diagnosis is usually clinical

Chlorpromazine jaundice is particularly difficult to exclude, because it is not rare for patients with non-specific upper alimentary tract symptoms to be given

chlorpromazine. Not long afterwards jaundice develops, and it is difficult to determine whether the patient has infectious hepatitis or not. Patients with chlorpromazine jaundice usually have a relatively high serum alkaline phosphatase level for their serum bilirubin level and little change is shown in the results of their flocculation tests, while the serum transaminase level is not particularly raised, eosinophilia in the peripheral blood is common and the serum cholesterol level is usually raised.

Course.

General and gastro-intestinal symptoms decrease with the onset of jaundice in most cases, so that symptoms are often minor when jaundice is at its peak. After two weeks the average patient feels reasonably well, except for lassitude, but liver tenderness and enlargement tend to persist for longer; indeed, liver tenderness may outlast jaundice.

The course of cholangiolitic hepatitis is usually identical with that of the more classical acute infectious hepatitis. Cholangiolitic hepatitis is a term applied to viral hepatitis in which the features more closely resemble those of obstructive jaundice than of parenchyma cell damage. There is no evidence that a different virus is involved, and it seems that the effect of the disease on cholangioles is more apparent in some cases than in others in which the effect is more on parenchymal cells, but there is little difference as far as the patient is concerned—the management and prognosis are the same.

Complications.

Estimates of the incidence of complications are inaccurate, particularly since few anicteric cases are recognized, but the vast majority of patients with acute infectious hepatitis are left with no residual damage; they are completely cured.

Relapses.

Relapses are common in both the icteric and the non-icteric forms, and have been estimated to occur in up to 20% of cases in some epidemics. They commonly occur when the patient returns to activity, but are not considered to occur later than six months after the acute attack. Probably 95% of relapsing patients make an ultimate complete recovery, and so should be treated in exactly the same manner as those in the acute primary attack.

Continuing Hepatitis.

Continuing hepatitis refers to acute infectious hepatitis which has lasted for more than four months. It is uncommon, but hardly rare. Neefe suggests that 15% to 20% of acute cases may fall into this group, but 95% of them still recover completely within 18 months. Improvement is usually gradual, and jaundice, fatigue and biochemical abnormalities persist. The serum transaminase level may be quite high—for example, 300 or 400 units. Liver function tests may not serve to distinguish between continuing hepatitis and progressive hepatitis, and liver biopsy may be necessary to make a correct diagnosis; there will be evidence of continuing inflammation, but no cirrhosis, fibrosis or significant necrosis and no alteration of the architecture. Whilst these patients make an ultimate recovery without permanent liver damage and restriction of activity is recommended, there is little doubt that the judicious use of adreno-corticosteroids may induce a more rapid improvement. However, steroid treatment even for a week or two is not without its short-term and long-term dangers, and should be used only after careful consideration of all the factors, particularly since the vast majority of these patients make an ultimate recovery without steroid therapy.

Progressive or Chronic Hepatitis.

Progressive hepatitis, which means continuing inflammatory disease of the liver with histological evidence of cirrhosis and grossly abnormal results from all liver function tests, does appear occasionally to follow upon an acute attack of hepatitis. More often it appears in patients who have no clear-cut history of acute infectious hepatitis preceding it. In some cases the condition becomes quiescent and stabilized, and an inactive state may persist through the years, but the disorder has a high mortality within five years of the acute onset from liver failure and other complications of cirrhosis. Adrenal corticosteroids and ACTH appear to benefit some patients, but not all; bed rest appears to be of benefit, but in some cases continuing activity has not prevented the development of an inactive disease state. An optimistic viewpoint appears justified unless the patient shows progressive diminution in liver function. It is a useful rule to prescribe rest and steroids when the patient is sick and the transaminase and bilirubin levels are high.

The Post-Hepatitis Syndrome.

Some patients continue to have minor symptoms of anorexia, fatigue and abdominal discomfort after recovering from acute hepatitis. They do not show altered results in liver function tests or continuing disease, but rather psychosomatic illness. A number of patients have "biliary" discomfort for a few weeks in the latter part of convalescence—a common and insignificant phenomenon.

Prognosis.

The vast majority of patients with hepatitis make a complete recovery without microscopic or biochemical evidence of residual hepatic damage. The average young adult with uncomplicated icteric hepatitis will be well in six weeks and will rarely be off work for more than three months. A prolonged course, or the development of a relapse, increases the chances of failing to make a complete recovery, but there is still very little difference in ultimate prognosis.

A fatal outcome has been reported to occur in one or two cases in each 1000. Some factors tending to increase the mortality are increasing age, the presence of other disease (especially of the liver), the general health of the patient, probably the strain of the virus, perhaps the size of the infecting dose and the management of the patient. A considerably increased mortality has been reported in girls at the menarche, in post-menopausal women, in older debilitated persons with other diseases and in patients with cirrhosis.

Death occurs in the fulminant case quite early, but in subacute cases it may be delayed for several weeks and occur in a sudden relapse, or as a part of the gradual progression of the disease. Death usually occurs from hepatic failure with coma.

It is important to emphasize that the prognosis cannot be judged by the initial severity of the acute attack or by the absence of jaundice; this refers to an acutely fatal outcome as well as to a liability to develop progressive hepatitis.

Prophylaxis.

Prophylaxis means prevention of contact with the infecting agent, which means, quite baldly, the prevention of fæcal material from reaching the mouth. This implies the prevention of fæcal contamination of fingers and food, the washing of hands and all the measures designed to prevent the spread of intestinal pathogens—that is, perfect hygiene. It is obviously desirable to avoid close and unnecessary contacts during an epidemic, because every person is a potential carrier.

As the virus is present in the blood, very great care should be taken in using syringes; and an epidemic of hepatitis provides yet another reason for being quite certain that blood transfusion is absolutely necessary.

Prophylaxis by the administration of 0·01 ml. of gamma globulin per pound of body weight is of real value in reducing the incidence of clinical disease, but it is not of value after the onset of the disease. It is desirable to give gamma globulin to all persons exposed in a spreading epidemic in an institution or a closed community and to all those living in close contact with those heavily exposed in any circumstances.

A patient treated in hospital is managed with the same strict precautions as are adopted for poliomyelitis, but one treated at home need not be isolated from close members of the family, as they have probably been exposed by the time the disease presents. Nevertheless, advice must be given to other members of the family, and to the patient, regarding strict personal and family hygiene.

Treatment.

Rest in Bed.

Bed rest is indicated for all patients, whether jaundiced or not, in the acute phases of the disease, and they should be so confined at least till they are clinically no longer ill. It is recommended that patients be kept at rest until significant tenderness and enlargement of the liver have subsided. The precise duration of bed rest varies from patient to patient and, in general, is more prolonged in severe cases and in patients over 40 years of age. It is common and good practice to keep patients at rest till they feel well and exhibit no significant signs, and jaundice has disappeared or is minimal. Although recent publications have suggested that bed rest may be of little value, it is well to remember that relapses during convalescence frequently occur after exertion or alcoholic excess, as was often noted in the Australian Army in the Middle East and elsewhere. Alcohol should be forbidden during the acute attack, during convalescence and for the next six months. It must be reemphasized that it is impossible to judge the course and outcome of acute infectious hepatitis by the initial presentation and severity of the acute attack.

Convalescence.

Convalescence should be gradual, and patients who are over 40 years of age, who have had severe attacks or who have had prolonged symptoms, should be warned to avoid excessive exertion and all alcohol for at least six months. However, it is important to avoid excessive restriction, as the "iatrogenic post-hepatitis syndrome" is common.

Diet.

A high-calorie, high-protein diet is the best, and fat should be restricted only if the patient is nauseated by it—a not uncommon complaint. During convalescence, minor dietetic alteration may decrease some of the complaints of right hypochondrium discomfort.

Drugs.

No drugs appear to be of significant value, and the side effects of the broad-spectrum antibiotics far outweigh their recorded benefits. Vitamin K, given orally or intravenously, is indicated for the patient who has significant bleeding. ACTH and cortisone are not indicated in ordinary acute hepatitis, and there is some evidence that relapses are more frequent in patients given them. These preparations should be reserved for patients with very severe hepatitis or hepatic coma.

Treatment of Coma.

Hepatic coma ushered in by lethargy, sleepiness, reversed sleep rhythm, disorientation and flapping tremor signifies that severe hepatitis is present, but not necessarily that death will ensue. These patients should be treated vigorously like other patients with hepatic coma. Large doses of steroids—that is, 40 of ACTH given intravenously or 100 mg. of prednisone given orally—each day, six grammes of neomycin daily, a diet free from protein and adequate fluids and electrolytes form the basis of management.

Symptomatic Measures.

Anorexia and Nausea.—Anorexia and nausea, which are often severe, may be relieved by "Benadryl" or "Marzine"; chlorpromazine is contraindicated. When anorexia and nausea are sufficiently severe, an infusion of a 10% dextrose solution may be needed each day. In such cases vitamin supplements are always given.

Pruritus.—Pruritus may be very troublesome for a few days, preventing sleep, and may be relieved by antihistaminics or small doses of pilocarpine; in a rare case intravenous administration of papaverine may be used. Great caution should be used when giving ergotamine, because serious accidents have occurred in patients with liver disease who were given this drug; doses should not be repeated with the frequency used for migraine.

Insomnia.—Barbiturates may be used for insomnia, but it is wise to give smaller-than-average doses until the effects have been observed. "Amytal", "Nembutal" (pentobarbital) and "Seconal" are mainly degraded by the liver and may have a greater effect than anticipated. Phenobarbital is excreted in part by the kidneys and is safer, if used in half-dosage.

Lipotropic Agents.—These are without value unless the patient has significant nutritional deficiencies, and they are not needed if the patient has a normal diet. Methionine may be dangerous if given in large amounts, since it may precipitate coma.

Follow-up.

It is desirable to see the recovered patient at monthly intervals at first and then at longer intervals for a year, especially if there has been evidence of persisting infection, to detect any early evidence of smouldering infection.

Conclusion.

Acute infectious hepatitis is transmitted by fæcal contamination from person to person in the majority of instances. Symptomless carriers and children are probably of very great importance. Rest is desirable for all patients until the course of their disease becomes clear, and apart from a high-calorie, high-protein diet, there is little evidence that any other form of treatment makes a significant contribution towards shortening the course of the disease. In complicated cases steroid treatment may be of real value. The vast majority of patients with acute infectious hepatitis completely recover and have no further evidence of the disease. It is important to differentiate acute infectious hepatitis, continuing hepatitis and progressive or chronic hepatitis, and these terms are literally correct. The disease is preventible and should be prevented.

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Special Article.

DOCTORS AND THEIR FAMILIES.

READERS of this journal may remember completing in June, 1960, an information sheet requiring their age group, and their father's, grandfather's and children's occupations. The information given has been analysed and certain inferences have been drawn from it. As was stated in the letter acompanying the information sheet, the data about medical men were to be used in a wider programme of research concerned mainly with the teaching profession. However, this article has been prepared in the hope that the data obtained may be of interest to those who were kind enough to cooperate by completing the sheet.

Scope of the Study and Procedure.

With the assistance of the New South Wales Branch of the British Medical Association, a letter of advice and an information sheet were sent to each male member of the Association in N.S.W. Details of replies received are as follows:

Number of doctors approached	3500
Number of replies received	2680 (76·6%)
Number of usable replies	2654 (75·8%)

The facts reported below are based, then, on approximately three-quarters of all male doctors in New South Wales who are members of the B.M.A. Whether the same

facts would be true of the one-quarter who did not reply, or of non-members, is, of course, not known. It would be a not unreasonable guess that they would be.

The Occupational Background of Doctors.

The occupations of the doctors' fathers, grandfathers and children were classified into six categories, which form a rough gradient in social esteem. An indication of the type of occupation allotted to each category is given below:

Category 1: Professional and high administrative—for example, doctors, lawyers, "substantial" graziers.

Category 2: Headmasters, school inspectors, clergymen, owners or directors of "substantial" retail businesses.

Category 3: Station managers, teachers, higher clerical positions, physiotherapists, etc.

Category 4: Small shop owners, small farmers, skilled tradesmen, commercial travellers, nurses, receptionists.

Category 5: Semi-skilled tradesmen, shop assistants, mine-workers, minor clerical and postal workers, shearers.

Category 6: Unskilled manual workers—cleaners, caretakers, labourers.

Table I shows the distribution of the doctors' fathers in the six categories. To assist in the interpretation of these figures, the distribution by occupations in the same six categories of the total male work force in N.S.W. is given.[1] These latter figures are for 1947, but will serve as a guide.

TABLE 1

Comparison of Occupation of Doctors' Fathers with Male Work Force in N.S.W.

Category.	Proportion of Doctors' Fathers.	Proportion of Male Employees.
1	26·5%	0·82%
2	23·5%	3·34%
3	15·3%	5·42%
4	25·9%	49·47%
5	8·0%	28·34%
6	0·6%	12·61%
Total number..	2654	946,516

Table I shows strikingly the highly selective entry to medicine when judged by parental occupation. Approximately half the doctors' fathers are drawn from categories 1 and 2, and these categories include approximately 4% of all male employees. To put this another way, one in every two doctors in N.S.W. has a father who is employed in a type of occupation that is engaged in by only four in every hundred men employed in N.S.W.

These figures are for the total sample of doctors. When this sample is analysed in age groups, two further facts appear: one, that the youngest doctors are even more selectively recruited than the figures above suggest; the other, that there has been a change in the social pattern of recruitment to medicine over the last 30 or 40 years. The figures in Table II will make this clear.

Australia is generally regarded as a country with a fairly high degree of occupational mobility. Its educational systems provide a generous measure of equality of opportunity. One might predict from this that there would be an increasing trend for recruits to professions such as medicine to be drawn from a wider section of the community. In fact, the reverse seems to be occurring in medicine, at least in N.S.W. The youngest doctors, when compared with the older members of the profession, are being drawn in significantly greater proportion from a more restricted social and occupational group in the community. This fact is corroborated by the figures for categories 3 and 4 combined, which show a progressive decrease in recruitment from these "lower" categories.

Some of the younger doctors would have been helped to enter medicine by the Commonwealth Reconstruction Training Scheme, and later by the Commonwealth Scholarship Scheme. If the Commonwealth Scholarship Scheme is having any effect in altering the pattern of home background of recruits to medicine, this effect would probably be best seen in the students at present in training. Unfortunately, results for this group are not available.

TABLE II.

Category.	Proportion of Doctors' Fathers by Age Group of Doctors.			
	51 Years and Over.	41 to 50 Years.	31 to 40 Years.	21 to 30 Years.
1 and 2 combined	41·0% (295)	52·3% (329)	53·1% (557)	58·2% (149)
3 and 4 combined	49·1% (353)	39·9% (251)	39·2% (412)	30·8% (79)

It is of interest to inquire whether this narrowing of the range of occupations from which doctors are drawn is due to an increasing recruitment from the homes of doctors themselves. In fact, this does not appear to be the explanation, although there has been a slight tendency for this to happen. This is seen particularly when the oldest and youngest members of the profession are compared. The percentages of doctors in each age group whose fathers were doctors are as follows: 51+ years, 12·8%; 41 to 50 years, 17·8%; 31 to 40 years, 16·8%; 21 to 30 years, 15·6%. The same fairly constant figures are seen in the percentages of doctors in each age group whose father and grandfather were both doctors. The figures are: 51+ years, 1·5%; 41 to 50 years, 1·9%; 31 to 40 years, 2·2%; 21 to 30 years, 2·0%. The increased recruitment from top-ranking occupational groups must be due mainly to the more general attraction that medicine as a career has to these groups, rather than to any increase in the tendency of the profession to reproduce its own kind.

The trend towards increasing selectivity in recruitment to medicine is seen clearly if the analysis is extended to include the grandfathers (paternal) of the doctors. A comparison between the proportion of fathers and grandfathers in each category is given in Table III.

TABLE III.

Category.	Proportion of Fathers.	Proportion of Grandfathers.
1	26·5%	9·8%
2	23·5%	18·2%
3	15·3%	12·1%
4	25·9%	45·0%
5	8·0%	12·4%
6	0·6%	2·5%
Total number	2654	2476[1]

[1] The discrepancy between the number of grandfathers and the number of fathers arises because some doctors did not know their grandfathers' occupation.

The upward social mobility is quite apparent from an inspection of Table III. Whereas about 50 in every 100 doctors come from a professional or similar type of home (categories 1 and 2 combined), only about 28 in every 100 of their fathers did so. The evidence from categories 4, 5 and 6 tells the same story in a different way. Nearly twice as many grandfathers as fathers of doctors were employed as skilled tradesmen and the

[1] "Census of the Commonwealth of Australia, 30th June, 1947. Part XVIII: Occupation." Published 1952; adapted from Table 4, pp. 1410-1501.

like, semi-skilled workmen and unskilled workmen. One is tempted to infer that the opportunity to improve one's occupational status, irrespective of occupation, was greater in the generation once removed, than in the last generation, which is a curious reversal of the view generally held that educational opportunities have been steadily improving.

The Occupations of Doctors' Children.

The occupations being followed by doctors' children were classified into the same six categories. The employment pattern for male and female children was so different that results have been kept separate. The distributions are given in Table IV.

TABLE IV.

Category.	Male Children.	Female Children.
1	58·3% (438)	6·7% (44)
2	7·1% (53)	1·2% (8)
3	17·2% (129)	46·6% (304)
4	15·6% (117)	45·0% (294)
5	1·6% (12)	0·5% (3)
6	0·3% (2)	0% (0)
Total number	751	653

The concentration of male children in categories I and 2 (65·4%) is remarkably high. As will be seen later it is accounted for largely by the high percentage of doctors' male children who themselves enter medicine. If the male line only is followed, and the comparison is based on category 1, the change from the doctors' grandfathers (9·8%) to the doctors' male children (58·3%) is striking. The magnitude of the figures can be appreciated by a comparison with teachers. Figures for two generations only of teachers are available. These show a movement from secondary school teachers' fathers (0·4%) to their sons (18·8%) in category 1.

The differences between the two professional groups cannot be explained on the information available. Educational opportunity, superior intelligence, vocational ambition, economic position and professional tradition would all play a part. The only comparative evidence on the intelligence of the two groups of children is the percentage of each group with a Leaving Certificate pass marking the end of the secondary school course — 86·2% for doctors' sons and 72·4% for secondary school teachers' sons. However, this evidence is very slight. It obscures the quality of the examination passes, and it obscures the economic factor which plays a part in making it possible for children to complete a secondary school course.

The employment pattern of doctors' daughters is very different, relatively few being found in the top professional positions. This tendency would probably be apparent in the children of any occupational group. The social status of women seems to be achieved largely through marriage.

The Continuity of Medicine as a Profession.

An intensive study of the continuity of medicine as a profession in families would require more information than was secured for this study. To the information gained, one would at least have to add facts about the doctors' siblings and maternal forebears. However, the facts available do yield some evidence of interest. This can be presented in a number of ways.

Of the 2654 doctors who replied, 51[1] (or 1·9%) had both father and grandfather who were doctors; 421[1] (or 15·9%) had a doctor father. If these results are combined, it could be said that 472[1] (or 17·8%) of the doctors studied had followed medicine as a profession for at least two generations. One needs comparable figures from other occupations in order to interpret these. One comparable measure is available in respect of teachers. Of a group of 2299 teachers studied, 8·4% had a teacher-father. It can be seen from this figure that it is almost twice as common for a doctor to have a doctor-father as it is for a teacher to have a teacher-father.

The degree of professional continuity can be examined also by determining the number of doctors' children who themselves become doctors (or are in training for medicine). Of the 751 male children in employment (or training), no less than 43·8 have entered medicine. (The comparable figure for teachers is 25%.)

When the daughter's occupations are examined, it is seen that while a considerably smaller proportion goes into medicine (4·6%), a very large percentage (46·7%) goes into allied occupations such as nursing and physiotherapy. If sons and daughters are taken together, and occupations allied to medicine are taken together with medicine, no less than 47·3% of doctors' children who have taken up a career are accounted for. Nearly one in every two employed children of doctors has entered medicine or an allied occupation.

The facts presented suggest a substantial professional continuity. Such continuity must confer many benefits on the profession. One would surmise that children of doctors who do medicine pursue their studies in an educational setting where the example, stimulation and assistance available to them from home would be very favourable. Professional ethics also, which must be a difficult "subject" to teach, could be more easily transmitted in a meaningful way. On the debit side, one wonders whether such a high degree of occupational exclusiveness could produce undue restriction of interest and cultural breadth.

The factors at work in determining the pattern of recruitment to medicine may well have significance for medical education, and for the wider role of the medical man in the community.

Addendum.

In lighter vein the following comments are added. The examination of the doctors' replies proved to be very interesting and often entertaining. A surprisingly large number of doctors did not spell "medicine" correctly. Statistics for this were not kept.

One doctor returned the sheet without the information sought, but with the stated promise that he would do so if I sent him a fee of ten guineas.

Some were not sure of what their grandfathers had done; others seemed proud of their unusual occupations. These produced a crop of delightful occupations, such as, "no hoper", "professional smuggler", "not sure, but he wasn't a very big noise", "opened coffee shop, tried to convert . . . [I shall leave out the town] to temperance, but in this was not successful", and "indolent recluse". Others, in describing their children, gave such answers as "domestic duties, now married, still domestic duties", "married, never gainfully employed", and "two delinquents aged 2½ and 4½".

I liked the touch provided by one doctor who sent the sheet back (completed) with the comment: "For further details see 'Who's Who'."

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[1] Considered as separate families, these figures would be inflated to an unknown degree by the fact that some of the doctors studied would be brothers.

Points of View.

WHY AN INSTITUTE FOR REGIONAL RESEARCH?

THE establishment of the Myall Valley Institute for Regional Research by the Tea Gardens Memorial Hospital Trust arises from the contention that national research should be organized on a regional basis, with a central supervising body. This would have the following advantages:

1. Each region has distinct research problems of its own, and regional research institutions would best serve their investigation.

2. By standardizing recording procedure the nationwide picture of a problem could be obtained with utmost economy and speed, being built up from regional results.

3. While portion of the activities of the regional institution would serve fact-finding for national purposes, a large measure of time could be left open for the pursuit of the individual research ideas of the regional research workers, so that the major impetus in research, namely, the cultivation of individual imagination and inspiration, is not lost, as could easily happen in a large, centralized organization.

4. Regional standardized storehouses of all information available about a region would greatly facilitate research.

5. Within a region the number of research workers is usually sufficiently small to allow close personal contact between all of them, fruitful discussion, mutual assistance and discovery of relationships between special fields unlikely to come in contact in the metropolis; e.g., a geologist recording special climatic data in the course of his work can make these available to someone doing morbidity studies. Something of the old spirit of *Universitas litterarum*, nowadays lost in the hustle and bustle of specialization, could thus be maintained.

6. Space for accommodation of scientists could ultimately be available in every region.

7. Decentralization of regional information would relieve the pressure on centralized libraries and institutions and is desirable from a strategic point of view.

8. Institutes for regional research could focus public attention on the importance of research in every region and make the regional centre and its achievements a matter of civic pride.

9. Youngsters with interest in the exploration of nature and science (potential future research talent) could be encouraged and helped at an early stage by close contact with actual investigation and fact finding close to their homes.

10. Lay groups interested in research, historical societies, National Park and Fauna groups, etc., could have a local centre for meetings, discussions, voluntary work and expert advice.

The Myall Valley Institute for Regional Research, being inspired and directed by a medical practitioner, will of course focus its attention on regional medical problems, predominantly general practice problems. This trend is emphasized by its administrative combination with a local accident service, an essential service in an isolated township without a general hospital and with a rapidly rising tourist influx. The Tea Gardens Memorial Hospital Trust has displayed remarkable initiative and vision in accepting control of the institution. Naturally, for a start, the facilities are limited. However, a start in what is felt is the right direction has been made, which will slowly but surely—it is hoped—add something of value to this region, this nation and mankind in general.

HANNS PACY,
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Myall Valley Institute
for Regional Research.

Tea Gardens,
New South Wales.

British Medical Association.

NEW SOUTH WALES BRANCH: SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on July 28, 1960, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. B. A. COOK, the President, in the chair.

Pre-Operative and Post-Operative Intravenous Therapy.

DR. FRANK FISHER read a paper on pre-operative and post-operative intravenous therapy. This paper has not been made available for publication.

DR. D. E. FAILES read a paper entitled "Pre-operative and Post-operative Intravenous Therapy" (see page 200).

DR. A. C. BOWRING, in opening the discussion, said that he was happy that the Section of Pædiatrics was represented at the meeting and glad to open the discussion on its behalf, since he felt that the problems under discussion, which were by and large an inexact science, had always been studied more intensively and more critically by pædiatricians than by their counterparts in adult spheres of medicine. Both speakers had pointed out that the more complex problems of pre-operative and post-operative fluid therapy could be adequately managed only in specialized institutions, and with that he would heartily agree. However, he would like to make the point that the great majority of fluid and electrolyte problems were simple ones, concerned only with the maintenance of an adequate supply of body water and constant osmotic pressure, and that they could be managed in most hospitals by anyone who was prepared to become familiar with the techniques and procedures involved. He thought that further attention to that type of fluid problem by a wider circle of the medical profession would result in an improvement in the standard of treatment of children generally. As those therapeutic substances were very dramatic in their benefit or harm, the need for adequate laboratory facilities in their supervision had been well and frequently stressed. However, he would like to stress that even more important in most cases was the need for frequent clinical reassessment, and that was particularly so in childhood. As the number of particles of the electrolyte was the important factor, they should have by this time abandoned description of their measures by gravimetric means, and the terms "milliosmole" and "milliequivalent" should have been widely adopted. He believed that the problems would be further simplified when they considered the patient's weight in kilograms and the composition of all fluids used for intravenous therapy in terms of milliequivalents per litre; moreover, they should measure body fluid losses in terms of millilitres rather than as ounces. The two speakers that evening had by and large adhered to those principles, but even so, one heard reference to pints of blood, patients weighing so many pounds, and requirements of so many grammes of salt. Before setting their arbitrary decisions in those matters beyond the patient's faculties for selective absorption, they should be very conscious of the patient's requirements at each weight and age, and of the significance of physical findings and laboratory tests in relation to the patient's age, weight, surface area and any other disease process. Only in that way could they avoid the errors which might occur in endeavouring to correct a serum sodium level of, for example, 120 milliequivalents per litre in a child suffering from compensated cardiac failure or pancreatic steatorrhœa who had compensated for his low sodium level and become ill only when an attempt was made to return it to a level which was normal for another person.

Dr. Bowring went on to say that Dr. Failes had stressed the desirability of collecting gastro-intestinal fluid lost over the 24-hour period and then performing electrolyte analyses on the specimen of fluid. Dr. Bowring commended that as being particularly valuable in the new-born, since in those patients the concentration of fluid lost from an intestinal obstruction might vary enormously, not only from patient to patient, but in any one patient from day to day. He did not think that one could generalize about the composition of such fluids and replace them by rule of thumb.

Dr. Bowring further said that at the meeting they could go extensively into the ways in which the problem in childhood differed from the general problem in adults, because they were so numerous. He thought it reasonable to state that in the child, fluid and electrolyte therapy was a more exact science, since, as reserves of those substances were related to body mass, and as turnover was related to body surface, the changes were more rapid and dramatic with the larger surface-mass ratio of small patients. The need to be familiar with the requirements of patients of various ages had been mentioned by Dr. Fisher. Further, in the management of those problems in childhood, special techniques of scalp vein infusion and the cutting down on veins for the insertion of cannulæ might require an expert in those procedures. However, he believed that everyone could become sufficiently adept to be able to find one vein into which fluid might be given to any child in an emergency. Probably the greatest barrier to the extension of more exact fluid therapy in childhood was the need for special skill and diligence by the nursing staff in the management of those problems. It was fatuous for medical attendants to estimate the child's requirements to the last millilitre if the nurse was not equally accurate in her observation and maintenance of the fluid balance chart. Thus, if a nurse allowed a child to vomit and did not change the bib, a second vomit occurring in that same bib would cause an inaccuracy

of perhaps 30 ml. in the estimated loss. The control of drip rates in small infants also appeared to be a problem which nurses could master in an extraordinary way, but only with considerable experience. In the neonatal period, the post-operative fluid management was greatly simplified if operation could be performed early after birth, so that the two or three days in which fluid and electrolyte requirements were minimal could be used in the immediate post-operative period during which the child might be suffering from ileus or some other source of fluid depletion. If operation was deferred until the third or fourth day, the child had a very much greater post-operative fluid problem. The significance of accurate measurement and replacement of blood loss had been well stressed by Dr. Fisher. Dr. Bowring said he thought it worthy of mention, particularly in the new-born period, that that was a "must" for anyone who operated on such children. A child weighing 3 kilograms, which was an average birth weight, had a blood volume of about 250 ml. To that child the loss of 100 ml. of blood could be a fatal hæmorrhage, and would correspond with a loss of some 2 litres in the average adult. He commended very strongly measures recommended by Dr. Fisher for weighing blood lost, and said that he had found that, with the adoption of that principle, one of the advantages was that surgical technique became slightly modified so that blood loss was greatly reduced. Finally, he suggested that all major intravenous fluid problems in childhood should be under the control of a pædiatrician. Those problems required teamwork, and whether the surgeon was a pædiatric surgeon or not, whether the hospital was a pædiatric hospital or not, he believed that by having the pædiatrician as the ultimate arbiter in the management of the intravenous therapy they would reach the optimum in treatment of the patient.

DR. L. T. SHEA said that he agreed with all three speakers. The subject was a difficult one, and the ground had been well covered. He had been pleased to hear Dr. Bowring say that a large percentage of cases were fairly simple, and fortunately for many practitioners nature was wonderful. Common sense was the main thing. He agreed with Dr. Bowring that for young children the pædiatrician was the man. On the other hand, he mistrusted many surgeons completely, in that, given an intestinal obstruction or something of the sort, they did not seem to be able to rid themselves of the belief that their operation was the thing, whereas many patients would not be saved by any operation while they had an uncorrected gross electrolyte disturbance. Surgeons should take a reasonable view of what those people required in the way of resuscitation. He thought that the average anæsthetist had better understanding of that than the average surgeon.

DR. JOHN BEVERIDGE said that in the papers presented he found much that was worthy of praise. For example, Dr. Failes should be congratulated on the emphasis he had placed on the history. Too often they tended to place great reliance on physical examination, and they did not pay enough attention to the history which gave them the clue to the mechanism whereby the disturbed state had been produced. Dr. Fisher should be congratulated on the emphasis he had placed on the basic physiology and its disturbances in their understanding of those problems. However, there were some things which also deserved criticism, and he hoped that those criticisms would be taken in good part. First, disturbances of water and electrolyte metabolism had been spoken of in the one breath. The dissection of those problems should be in terms of water upsets and electrolyte upsets. It was true that Dr. Fisher had referred to disturbances of osmotic pressure, but little attention had been paid other than that passing reference. If more attention was paid to disturbances of osmotic pressure, it would become clear that "normal saline", which had been recommended as replacement fluid, was an unsuitable fluid. Dr. Beveridge said that he could not imagine any fluid more unphysiological than "physiological saline". A fluid with such a concentration of sodium and chloride must rarely be necessary in clinical practice, and the use of hypertonic saline must be confined to excessively rare cases. Water intoxication, usually iatrogenic, might be one such indication. Secondly, it was disappointing that so little stress had been placed on disturbances of acid-base metabolism. Darrow's solution had been recommended as a replacement fluid when potassium was indicated; but it should be realized that Darrow's solution was a physiologically alkaline solution designed for replacement therapy in gastro-enteritis. An example of the loose thinking which surrounded the question of acid-base disturbance was the statement by Dr. Failes that in pyloric stenosis the loss of chloride ion was the most important loss. Although loss of chloride ion was important, the loss of hydrogen ion was by far the most important loss in pyloric stenosis, and it was that loss of hydrogen ion which produced metabolic alkalæmia. To suggest that the reversal of that alkalæmia by large amounts of "normal saline" was optimum treatment further deserved censure; there was very good experimental and practical evidence that the use of potassium chloride in combination with hypotonic sodium chloride solutions achieved reversal much more effectively. Finally, Dr. Beveridge asked two questions. The first was whether they thought it wise to prevent potassium deficiency from developing by adding potassium to the intravenous fluid infusions of all patients receiving fluids parenterally, once they had passed urine. The second was what they thought of alcohol given intravenously as a source of calories.

DR. T. E. WILSON said that he did not speak as an average surgeon—no one would admit to being that. He referred first to Dr. Failes' remarks on the need to keep up with the blood loss at operation, and said that he thought it an advantage to anticipate such blood loss. Officially, an amount of 1500 ml. was held to be the average blood loss in a pelvic exenteration; he thought it an under-estimate. In a pelvic exenteration and similar major procedures, the anæsthetist's duty was to be ahead of the surgeon and establish positive replacement. Dr. Wilson then referred to weight loss, and said that Wangensteen's book had a picture of an adult patient being weighed before and after operation. It was practicable, though costly and troublesome. In post-operative care geriatric patients had the same type of problems as pædiatric patients. Most geriatric patients received over-replacement of fluid. Dr. Wilson disagreed with Dr. Failes in the treatment of those patients, and he considered that it was not better to give a little more than a little less than calculated. In geriatric patients over-infusion of fluid was likely to produce pulmonary œdema. If the levels of any of the ions were slightly decreased, care was also necessary to avoid over-replacement. Dr. Wilson finally asked the speakers to comment further on geriatric patients.

Dr. Fisher, in reply, said that he agreed entirely with everything that Dr. Beveridge had said. A little intravenous alcohol was of great benefit to everybody. He had seen it used extensively in England to provide calories, and it was useful as a central stimulant. He could not agree so readily with the idea of forestalling potassium deficiency. He thought that the intravenous administration of potassium ions by everyone would soon lead to a great number of fatalities. In a specialist unit, where everybody knew what he was doing, it might do no harm. In prolonged intravenous therapy (more than 48 hours), particularly with operations in which paralytic ileus was a danger, it was worth while to give potassium. However, as a rule he disagreed with its administration. In reply to Dr. Wilson, Dr. Fisher said that he himself had made it clear that one should keep up with the blood loss at the same time and the same rate as it occurred. He entirely agreed that if one could keep 200 ml. of blood ahead, it was absolutely right to do so. It was also important to know that if a bottle of blood was broken during the operation, one was a little ahead and so it did not matter. Dr. Wilson had contradicted himself about geriatric patients. Dr. Fisher did not agree with him, but with Dr. Failes; it was very rare for a geriatric patient to get pulmonary œdema on the operating table. However, if treatment continued for many days, it could happen. Dr. Fisher entirely agreed with the attitude that one must keep ahead of the blood loss. He said that the figures he had given were from a series of well over 1000 cases in London. Dr. Wilson had referred to the problem of anæsthesia in geriatrics. Dr. Fisher thought it badly managed on the average; in some ways it was more important than pædiatric anæsthesia. It was essential to keep up with the blood loss as it occurred. Failure to do so could lead to the situation in which the relatives commented that "Dad hasn't been the same since he had his operation" and investigation showed that they were right. The deterioration must be related to any periods of operative anoxia and hypotension.

Dr. Failes, in reply, said that he could not completely agree with Dr. Beveridge's remarks. He himself had been considering the problem from the point of view of an adult patient, whereas Dr. Beveridge was considering the needs of the child. Dr. Beveridge had pointed out that children did not tolerate concentrated electrolyte solutions well, and that precise replacement of electrolytes was necessary in children. Whilst he agreed with those statements, Dr. Failes said he believed that in adults concentrated solutions were well tolerated and were in fact indicated in certain circumstances. For example, a patient with a chronic electrolyte loss from a fistula could develop a state of severe sodium depletion; in such a case it was rational and effective to give hypertonic saline, rather than to give enormous amounts of dilute saline solutions.

With regard to the problem of pyloric stenosis, Dr. Failes did not consider that potassium chloride alone would be adequate, since there was usually an associated sodium deficit, due to losses of sodium in the gastric mucus. Thus the recommended replacement fluid was normal saline with added potassium chloride. In general, the dangers of intravenous administration of potassium need not be overstressed; nevertheless, he did not advise that potassium be given intravenously as a routine procedure for the first 48 hours after operation, since it was not usually necessary during this period. By that time the patient was normally taking fluids by mouth, and thus the problem of intravenous administration did not then arise. However, if the patient was not doing well after 48 hours, he should be thoroughly assessed and might well require potassium under those circumstances. With regard to geriatric patients, Dr. Failes said that he agreed with Dr. Wilson that they required more careful consideration than the ordinary healthy adult. Deficits of fluid and electrolytes might be replaced by the usual solutions, but special care was necessary with regard to the rate of intravenous administration.

Dr. Cook, from the chair, said that the papers and discussion had been stimulating and provocative. The diseases seemed to have been divided into three categories—the disease *per se*, anæsthetic diseases and operation diseases—which were three distinct entities. It appeared to him that anæsthesia had been made so safe for the patient that many operations were now done that should not be done particularly in certain types of malignant disease, in which the end results were still tragically the same as they would have been if no operation had been performed. Some people said that the patient had been made safe for surgery; but the advance had not been sufficient to make surgery safe for the patient.

Out of the Past.

THE BUBONIC PLAGUE.[1]

[From the *Australasian Medical Gazette*, May 20, 1902.]

WE regret to report that the bubonic plague still continues in an epidemic form in some of the large cities of the Commonwealth. In Sydney the number of cases in the present outbreak up to date is 128: of these 36 have proved fatal. It would appear as if the infection among the rats was widespread, since every now and then we read of plague-infected rats being discovered in different parts of the city and suburbs. Dr. Ashburton Thompson has pointed out that the suburban councils have not acted with the vigor the occasion requires, and as one proof of this may be taken the report of the sanitary inspector in Woollahra, who recently found 48 out of about 290 houses inspected to be swarming with rats. Unless some more stringent regulations are enforced, and some severe punishment inflicted for rats being found on any premises, cases of plague will continue to occur in our midst, and the deaths will be laid at the doors of the citizens who are too indifferent to the health of their fellow citizens to lay a little rat poison or attempt to catch the rats in traps. We have to thank the officials of the Board of Health for escaping, so far, a very severe epidemic of the disease, for if it were not for their watchful care over the insanitary areas of the city, and the prompt measures resorted to, a much larger number of cases would have occurred.

A new feature in the epidemic during the past month has been the deaths of several animals at the Zoological Gardens from plague. The exact path of infection has not been clearly traced, but it is probable that the plague-infected rats have wandered from the Moore Park "tips" to the Gardens. During the cleansing operations at the Gardens some dead rats were discovered under the flooring of one of the houses. On the announcement of the outbreak of plague at the Sydney "Zoo", Dr. Gresswell, the Chairman of the Board of Health in Melbourne, visited the "Zoo" in that city, and, as a result, the Zoological Society was informed that, in his opinion, attention should be directed to the rectification of unwholesome conditions, and in particular to the extermination of rodent vermin. . . .

The disease still continues prevalent in Brisbane, and we regret to record the death of Dr. Wray, the Medical Officer of Health, after a few days' illness from it. We also regret to learn that Dr. Maclean, the senior resident medical officer at the Brisbane Hospital, is suffering from the disease, and is in a precarious state. It is said that in his case the symptoms developed rather suddenly after examining a patient suspected of suffering from the disease in the out-patient department of the hospital. In consequence of the prevalence of the disease in the large cities, it is only to be expected that stray cases will occur in the country districts. One undoubted case has occurred in Bowra, a small town on one of the Northern rivers of New South Wales. Country practitioners should therefore be on the alert when called to any case of sudden serious illness in a person who has recently arrived from a plague infected area.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

AN AUSTRALIAN MEDICAL ASSOCIATION.

SIR: It is my earnest hope that the close attention of all the members of our great association, entitled "The British Medical Association (*Australia*)", has been given to the Prime Minister's Australia Day message on January 26. It included these inspiring words: "The name 'Australia' is one in which we should have an overwhelming pride." That pride, I believe, would be manifested by an overwhelming majority in favour of the title "The Medical Association of Australia", were a referendum of the members to be held on the issue whether that title or that of "The Australian Medical Association" should be given to the new Association.

Yours, etc.,

North Terrace,
Adelaide,
South Australia.
Undated.

H. S. NEWLAND.

LONG-ACTING SULPHONAMIDES IN GONORRHŒA.

SIR: Since the introduction of long-acting sulphonamides, many claims have been made concerning their virtues. It is well known that the more rapidly excreted sulphonamides now have little effect on most strains of gonococci. Nevertheless, it was decided to try out clinically the effect of the more recently introduced long-acting sulphonamides on the local gonococcal strains.

A clinical trial was conducted on a total of 25 cases of acute gonococcal urethritis in males. The sulphonamides used were: sulphamethoxypyridazine, 18 cases; sulphadimethoxine, 3 cases; sulphaphenazol, 4 cases. In the dosages recommended, sulphaphenazol (2 tablets night and morning for 5 days), sulphadimethoxine (1 tablet night and morning for 6 days), sulphamethoxypyridazine (2 tablets at once, 1 daily for 5 more days), all failed to cure, and the urethral discharge continued unabated.

With these facts in mind, after treating 12 cases it was thought that no good would come of the test, as the gonococci were not reduced by the treatment, controlled microscopically.

To stress the futility of the use of these drugs in the treatment of gonorrhœa, sulphamethoxypyridazine was used in a further 13 cases of acute gonorrhœal urethritis. Nine cases were given 4 tablets at once, 2 daily for 5 more days; 4 cases were given 4 tablets daily for 6 days. Only when the recommended dose was quadrupled were two cases assessed as cured. Side-effects of headache and rash occurred in all cases on the quadruple dosage.

It is evident that these sulphonamides are ineffective against the locally prevalent gonococcal strains.

Yours, etc.,

Government Clinic,
136-138 Gertrude Street,
Fitzroy, Victoria.
January 27, 1961.

F. W. WILLIAMS,
Medical Officer.

MEDICAL MORBIDITY IN A GENERAL HOSPITAL.

SIR: At the risk of straying a little from the subject of medical morbidity in a general hospital, may I be allowed an opportunity to answer Dr. Noble's letter (MED. J. AUST., January 21, 1961)?

I feel that the word "geriatrics" is used in various senses, and there are at least two issues involved here. One is the practical problem of getting someone to deal with the elderly long-stay hospital patient. There is no doubt that this has provided the impetus which has led to the establishment of geriatrics as a specialty in such countries as Great Britain. Dr. Noble in effect acknowledges this when he describes the geriatric unit being established in south Brisbane, using the very name of a specialty which he denies has real existence. Dr. Sheldon of Wolverhampton described this specialty to me as having 10% scientific and 90% practical justification.

In my original letter (MED. J. AUST., November 12, 1960) I stated that a geriatrician should be a general physician. Of course the reverse should also be true, and I deplore, along with Professor Saint, the arbitrary cleavage of geriatrics and general medicine. No one with experience in general practice would feel the need for a new class of consultant geriatrician, and I would agree that there is a poor case to be made out for the establishment of privately practising consultants in this field. But it is incorrect to deny that there are any special problems associated with the aging process. One would hardly expect a physician to practise privately as a consultant in infectious diseases, nor as a consultant medical historian, yet these are fascinating fields for study. In a similar manner I regard geriatrics, used in the sense of meaning the study of the disease processes of old age, as an interesting and rewarding field of medicine, and as an entity.

I would also challenge Dr. Nobles statement concerning the changes found in old people and their cause. These changes may indeed be physiological rather than pathological; but until more is known about the aging process, it is premature to be dogmatic. The only way to find out is to search for the answers. When the answers are found, we shall be able to do something about the prevention of diseases which lead to long-term illness. This calls for research—research stimulated by the concept that the study of the aging process is an entity. Positive results would be most fruitful in preventing human suffering, or in terms of financial saving to the community, or even from the coldly intellectual aspect of the search for knowledge.

Yours, etc.,

Greenvale Village for the Aged, Victoria.
January 27, 1961.

DAVID C. WALLACE, Geriatrician.

DEATH CERTIFICATES: AMENDING LEGISLATION.

SIR: At the present time it is the practice of medical practitioners to frequently issue certificates of death in cases where they have not seen the patient in a period of three months immediately prior to the death and where no other doctor has so seen the patient.

It is, therefore, of considerable importance for medical practitioners in New South Wales to be aware that the *Coroners Act*, No. 2 of 1960, repeals the *Coroners Act* No. 36 of 1912 and other Acts, and amends the provisions of Section 27A of the *Registration of Births, Deaths and Marriages Act* of 1899, as amended by subsequent Acts. The effect of the amendment is that medical practitioners may not issue certificates of death in the circumstances mentioned in the preceding paragraph, but shall, as soon as possible after the death, report such death to the officer in charge of the police station nearest to the place where the death occurred. The changed procedure will be effective from February 1, 1961.

Yours, etc.,

Department of Public Health, 52 Bridge Street, Sydney.
January 27, 1961.

G. R. G. CAMERON, Under Secretary.

ANATOMICAL FACTORS IN OCCUPATIONAL TRAUMA.

SIR: I read with considerable interest the above article by J. W. Perrott, in the issue of your Journal dated January 21, 1961. The author repeatedly stresses the need for careful observation of the patients' employment in order to arrive at a decision as to the influence of occupation in the causation of certain types of traumatic injuries.

Last year, the Division of Occupational Health visited large numbers of factories where there had been numerous cases of muscular strains and sprains, back injuries and tenosynovitis, etc. In many instances these factories employed an industrial medical officer, but unfortunately, few had visited the departments where these patients worked. When arrangements were made for joint inspections by the factory doctor, safety officer and ourselves, in many instances it was agreed that the manual handling methods were obviously unsatisfactory. In some cases it was possible to alter the process, often without much difficulty, thereby considerably reducing the likelihood of further trouble.

The number of factories employing either a full-time or part-time doctor is very small; only on rare occasions is a doctor able to visit a factory with which he has no connexion. If any practitioner desires a report upon his patient's working conditions, to enable him to arrive at a diagnosis, the Division would be pleased to make the necessary inquiries on his behalf.

Finally, for the information of those interested in this aspect of occupational medicine, may I draw their attention to the fact that during the last year my colleague, Mr. N. J. C. Peres, M.Sc., has published several articles in the *Australian Factory* on this subject; others have been accepted for publication in the near future.

Yours, etc.,

Division of Occupational Health, N.S.W. Department of Public Health, 86-88 George Street North, Sydney.
January 26, 1961.

ALAN BELL, Director.

PROFESSORSHIPS OF "ANÆSTHESIA".

SIR: Far from being pedantic, the observations of Dr. Marshall (January 21, 1961), for a distinguished dispenser of senselessness, are eminently sensible.

Licence in terminology is becoming widely accepted by the medical profession. But how can a department be pathological? What odd malady continuously bedevils it? Should not the walls of a varicose clinic be festooned with valveless veins in the exciting contemporary manner? And what future does a cardiac clinic hold for any brave but irregular heart?

Yours, etc.,

Newcastle.
January 27, 1961.

JOHN BROUGHTON.

SIR: Let us be realistic. There are over one thousand "chairs of anæsthesia" in Sydney today; you will find one in every dental surgery for a start.

If our English colleagues have strayed from the semantic path, need we follow them? There are dozens of sciences, and medical disciplines, with the suffix "ology", which has an exact meaning. Our American friends have appreciated this, and given us the word "Anæsthesiology"—"the science of anæsthesia and anæsthetics". We need not haggle over its length or its seven syllables. Others just as long are in common use—for instance, gastroenterology.

Yours, etc.,

British Medical Association House, 137 Macquarie Street, Sydney.
January 31, 1961.

COTTER HARVEY.

DEVELOPMENT OF THE OUTBACK MEDICAL SERVICES.

SIR: Dr. Russell Jones' letter (January 7) on the Royal Flying Doctor Service of New South Wales in reply to mine (November 19) is interesting rather than practical. However, he does back me up on the most important point of all—i.e., the need for expansion of the medical aspect of the Service. He even goes so far as to suggest two more flying doctors for New South Wales.

As I was five years in the pay of the service, he must assume that I am not unfamiliar with the costs and other

difficulties of what I propose, and not likely to "have a stroke" at anything which may be revealed in this regard. Indeed, it is not the amount of money spent that disturbs me. It is the way it is spent. I complained of the £20,000 a year spent on the upkeep of two aircraft and two pilots, because it is out of proportion to the £3000 spent on medical services.

There are two pilots at Broken Hill on full salaries, plus superannuation and other amenities, and not enough work for two. For instance, whilst the doctor is busy all day at a clinic, the pilot sits about with nothing to do, waiting to fly the doctor home that evening or next day. I have heard the opinion expressed out there, and I endorse it, that it would be better for the service to charter a pilot as required, as is done in Queensland, South Australia, Alice Springs and parts of Western Australia.

Each State runs its own service independently. Broken Hill Base flies, say, 60,000 miles a year at a maximum. At a charter rate of 2s. a mile, this would cost £6000. Add £4000 to cover return flights from clinics, and you have £10,000 compared to the £20,000 now spent.

Dr. Russell Jones agrees with me that liaison between the field or "network" and the administration in Sydney is bad. The secretary or manager (or both) should be resident in Broken Hill, not in Sydney, and an executive committee established there in liaison with the council in Sydney. They would then be close to the opinions and the convenience of the most important people concerned—i.e., the people of the network. Moreover, the bringing of delegates from the outback to council meetings in Sydney must cost the service £1000 annually, on top of much other travelling expenditure.

Queensland, a less wealthy State than New South Wales, spends a little more than double the New South Wales expenditure; but the money is spent to better purpose. Queensland supports three bases and four doctors to our one base and one doctor.

I stand firmly behind the principle that this is a flying doctor service, not an aerial ambulance service. That is what I believe John Flynn meant it to be. It is certainly what the people out there want.

Dr. Russell Jones states, that doctors are cheap compared to aircraft. A good doctor is more difficult to get than either aicraft or pilots, and more difficult to hold on to. During my time at Broken Hill, there was a chronic shortage of hospital residents at the Broken Hill Hospital. The same applies to most country hospitals.

By a good flying doctor, I mean one mature in experience and interested in that type of practice. He needs the qualifications of a good country general practitioner and little else, beyond some instruction in dental extractions and a love of flying. The idea that he should be young and venturesome is a myth believed in by lay councillors who are more interested in raising funds than in good doctoring. Certainly the sense of adventure may appeal to a young man; but after six or twelve months it wears off, and he becomes restless.

The life itself is a good deal less strenuous than ordinary general practice. Its interests and pleasures should be emphasized. Obviously, Dr. Russell Jones enjoyed it. He was widely known and respected, even though he was only there for eighteen months. There are no nicer people to work amongst than those outback, and I know of nothing more pleasant than flying to a clinic in the early morning stillness with an hour or two to enjoy the beauty of the landscape.

The value and the interest of clinics at outback places cannot be over-stressed. They are as important as any other function of the service. It is to be hoped that the council will eventually learn to understand more about them, and to realize the necessity for relieving the doctor of responsibility for emergency calls on clinic days. At present, if an accident occurs maybe 300 miles away, he is expected to drop everything, dismiss the waiting clinic patients, some of whom may have driven 50 miles or more to see him, and disappear into the blue.

I carried on clinics at all the places mentioned by Dr. Russell Jones for three years. After that, the new base at Port Augusta took over Andamooka and Marree, and I was able to turn my attention to the Far North-East instead of to the Far West. The Bourke area was served by Nancy Bird with her aerial ambulance work for the Bush Nursing Service in the 1930's. (There indeed was a romantic and venturesome enterprise! She used to land her light aircraft where she could. There were few airstrips in those days.) When the Flying Doctor Service started at Broken Hill in 1939, the Bourke area came to rely more on motor ambulance transport, and has never been in close touch with the service at Broken Hill. I started a clinic at Wanaaring in the hope of improving contact. What is really wanted, in my opinion, is another R.F.D.S. base at Bourke. Bourke has a good airport, and would make as good a base as any of the Queensland bases at Cloncurry, Charters Towers and Charleville. It would also serve a large area of country east of the Darling.

Finally, as regards night flying, Dr. Russell Jones mentions two occasions and says the experience cured both him and the pilot of wanting to do more. Such a statement shows up something fundamentally wrong with the organization. Momentous decisions like that, which affect hundreds of homes, should not be the responsibility of paid employees of the service, nor even of a couple of men on the executive in Sydney, who are, after all, amateurs as regards flying. Nor, indeed is the Department of Civil Aviation interested in pioneering matters that are primarily medical. This is an age of flying by night as well as by day, and it is an age of blood transfusions. When an emergency occurs at night in the outback, it is in most cases a matter of getting blood to the patient and getting it there quickly. The only occasion I was able to do this was before the Zinc Corporation removed their twin-engine Lockheed aircraft and their two pilots from Broken Hill to North Queensland. On that occasion, the patient owed her life to the Zinc Corporation rather than to the Royal Flying Doctor Service.

I do not advocate night flying with things as they are. It is too hazardous. What I do urge is the crying need for progress and pioneering in this direction, with the widest circle of technical advice. Progress, however gradual, should be made towards the elimination of hazards and towards the use at night of a few airstrips, strategically placed around the network, each one within reasonable motoring distance of a group of homesteads.

Yours, etc.,

C. R. R. HUXTABLE.

5 Warwick Street,
Killara,
New South Wales.
January 25, 1961.

PEPTIC ULCER: HERPES SIMPLEX AN ÆTIOLOGICAL AGENT?

SIR: Many attempts have been made to explain the cause of peptic ulcer. Avery Jones (1957)[1] in his Lumleian Lectures summarized many of the known facts, but the possibility of an infectious agent as a cause is not mentioned. Scant, if any, mention is given to the subject in most modern textbooks of medicine. Rosenow (1916-1923)[2] presented experimental evidence suggesting that streptococci might be responsible, but his work was not confirmed. Since then interest in an infectious agent as a cause seems to have waned, and very little has been written on this aspect in the English literature in recent years.

Certain comparisons between peptic ulcer and recurrent herpes simplex virus infection can be drawn. Both diseases are world wide in distribution and affect all ages and both sexes. Clinical observation suggests that, while both lesions do occur in childhood, the incidence of both increases in adolescence and adult life. They are both recurrent lesions, and recurrences generally occur in the same place. Recurrences may be precipitated in each case by known stimuli, such as trauma, infection, changes in environment, or in the physiological state of the body, emotional disturbances, etc. On other occasions, recurrences occur without known stimulus. Both diseases tend to be manifest as single, roughly circular lesions of about the same size, though multiple lesions are known in both instances. The initial stage of a recurrent herpes simplex lesion is vesicular: there is no report of a vesicular stage in acute peptic ulcer, though this is not unexpected, as maceration and peptic digestion would quickly break down a vesicular lesion into a shallow ulcer.

Herpes simplex virus is known to be capable of infecting many tissues in man, particularly those of ectodermal origin. Lesions on the lip, cornea, conjunctiva, buccal mucosa and genitalia are well recognized. Acute lesions have been reported in the œsophagus of children dying of disseminated disease, but apparently not elsewhere in the gastro-intestinal tract. Recurrent lesions do not seem to

[1] *Brit. med. J.*, 1957, 1: 719.
[2] *J. Infect. Dis.*, 1916, 19: 333; 1923, 33: 248.

be recognized below the pharynx. An herpetic lesion in the gastric or duodenal mucosa which was subsequently exposed to peptic digestion would, however, adequately explain many of the features of peptic ulcer.

This hypothesis has been the subject of attempted investigation. Two major avenues of investigation are apparent. One is to isolate herpes simplex virus from the gastro-intestinal tract of peptic ulcer patients, or better still from ulcers removed at operation, and compare these findings with those in patients without ulcers. Numerous viruses can be isolated from the gastro-intestinal tract, but their presence does not imply causal relationship to any disease process present. Herpes simplex virus can sometimes be isolated from the saliva of asymptomatic carriers. Scott (1959)[3] states that "isolation of the virus from patients with other clinical pictures must be reviewed in the light of a possible superinfection of damaged tissue by this ubiquitous virus".

The second method of investigation is to make a survey of peptic ulcer patients for the presence of herpes simplex antibodies, and to compare the incidence with that in non-ulcer patients. Antibodies are known to be present in a high percentage of the community, the percentage varying according to age and socio-economic status (Burnet and Williams, 1939).[4] This presumably reflects varying exposure to infection. Large numbers of patients and controls carefully matched for age, sex, social status and occupation might be necessary to provide an answer by this method.

Early in 1960, a pilot survey of the extent of herpes simplex neutralizing antibodies in peptic ulcer patients and controls was carried out by Miss K. Nolan, B.Sc., of the Queensland Institute of Medical Research. The method used was to incubate, at 37° C. for 60 minutes, inactivated serum diluted 1 in 5 in physiological saline with an amount of herpes virus known from previous titration to produce complete degeneration of HeLa cultures in three days, and then to inoculate each virus-serum mixture into two tubes of HeLa cells.

Of 23 patients with radiologically proven peptic ulcer 21 had neutralizing antibodies in their serum. Of 29 controls matched with ulcer patients by age and sex 22 also had neutralizing antibodies. The controls were hospital patients and laboratory staff, who were not questioned in relation to either peptic ulcer or herpes infection. The difference between these two rates does not reach the level of statistical significance ($P = 0{\cdot}2$).

As pressure of other work at the Queensland Institute of Medical Research has prevented further work on this project, and further testing is not immediately possible, it has been decided to present this hypothesis as a preliminary report in the hope that other centres might be interested and able to follow it up.

If this hypothesis is correct, acute ulcers in the gastro-intestinal tract might be expected to be relatively common and of equal incidence in both sexes. This is in accord with known experience of acute ulcers (Avery Jones, 1957). They cause few symptoms. Bleeding or perforation brings them to notice; they may explain many brief episodes of upper abdominal pain. In general, they probably heal quickly without trace.

Chronic ulcers must arise from acute ulcers. Pathologically, the herpetic lesion is one of necrosis. A necrotic lesion exposing muscularis mucosæ or deeper tissues to peptic digestion will result in an acute inflammatory reaction in these tissues, and will convert the ulcer from an acute specific (herpetic) one to a non-specific one. Thereafter the natural history of the ulcer will depend on the balance between the forces of destruction and repair. Fibrosis, thrombosis of vessels and adherence to neighbouring structures will encourage chronicity. Gastric secretion is necessary for chronic ulceration to occur, and in the case of duodenal ulceration, gastric hypersecretion is necessary to overcome the normally alkaline duodenal contents. The increasing incidence of duodenal ulceration in the community may well be due to factors in the diet or environment causing gastric hypersecretion.

The help and cooperation of Dr. R. Doherty of the Queensland Institute of Medical Research is gratefully acknowledged. The decision to publish this hypothesis in its present unproven state has been largely determined by the desire not to trespass further on his good offices. It is hoped to continue these investigations later. In the meantime perhaps someone else may be able to confirm or deny the validity of this hypothesis.

Yours, etc.,

Brisbane Hospital,
Brisbane.
January 5, 1961.

A. F. KNYVETT.

[3] "Viral and Rickettsial Infections in Man", 3rd edition, Pitman Medical, London, 1959: 758.

[4] MED. J. AUST., 1939, 1: 637.

RESUSCITATION IN APPARENT DROWNING.

SIR: In view of the recent publicity given to the mouth-to-mouth method of resuscitation in the apparently drowned, I would like to draw attention to a rough classification of deaths from drowning which I have seen in my post-mortem examinations as pathologist for the local coroners over the past seven years. There are three rough divisions.

1. The bronchi are filled right up to the bifurcation of the trachea with frothy, watery, blood-stained fluid—these cases usually come from the ordinary, fairly shallow swimming pools. In these cases, particularly, I believe that the mouth-to-mouth and/or mechanical air or oxygen resuscitation apparatus are doomed to failure unless some method is used to remove the intrabronchial fluid by, say, postural drainage and/or chest pressure, using the Holger-Neilsen method or the Eve rocker method.

2. There is very little water in the lungs, and these cases usually come from deeper pools. In these the main factor is probably some nervous inhibition or shock from cold, or, what I think frequently happens, is that one child jumps or dives onto another, producing unconsciousness. These cases, if they respond at all, would do best with oxygen apparatus.

3. There are the cases from rivers, where there is a number of factors operating, such as cold, and there are all sorts of debris in the bronchi, sticks, leaves, gravel, and some frothy fluid, but not as much as in the first group. Here I think there is mechanical irritation as well as inhaled water.

Recently I was present at a near-drowning, and, on suggesting postural drainage, i.e., to let the patient lie with his head down the slope of the bank, I was told by ambulance officers with a machine that postural drainage was "out of date and unnecessary".

The investigations done in Sydney on the mouth/nose method are very creditable, but it must be remembered that they were done on patients with dry lungs.

Would it be possible, please, to open up a discussion in your columns, particularly from pathologists who have dissected the drowned, in the hope that a better approach, which can be taught to first-aiders and other lay people, may eventuate,

My examinations have all been on fresh-water drowning cases.

Yours, etc.,

303 McCrae Street,
Bendigo,
Victoria.
January 30, 1961.

W. STRAEDE.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course in Neurology.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course in neurology will be held in the Students' Lecture Room of the Royal North Shore Hospital of Sydney on Saturday and Sunday, March 11 and 12, 1961. The course will be under the supervision of Dr. George Selby, and the programme will be as follows:

Saturday, March 11: Symposium on cerebrovascular disease: 2 p.m., "The Anatomy and Pathology of the Intracranial Vessels and Hæmodynamics of the Cerebral Circulation", Dr. Brian Turner; 2.30 p.m., "Cerebral Thrombosis—Diagnosis and Management", Dr. Eric Davis; 3 p.m., "Cerebral Hæmorrhage—Diagnosis and Management", Dr. W. Wolfenden; 4 p.m., "Insufficiency of the Carotid Circulation", Dr. W. J. G. Burke; 4.30 p.m., "Insufficiency of the Vertebral-Basilar Circulation", Dr. John Allsop; 5 p.m., "Radiological Diagnosis of Cerebral Vascular Lesions", Dr. Peter Cahill.

Sunday, March 12: 9.30 a.m., "The Surgical Management of Intracranial Hæmorrhage", Mr. W. Lister Reid; 10.15 a.m.,

"The Surgical Treatment of Extracranial Vascular Lesions", Mr. John M. F. Grant; 11.30 a.m., case demonstration; 2 p.m., "A Review of the Myopathies", Dr. K. B. Noad; 2.45 p.m., "The Indications and Value of Electromyography in Neurological Diagnosis", Dr. R. W. Johnston; 4 p.m., "Observations on Kuru" (with colour film), Dr. L. R. Rail; 4.30 p.m., quiz session.

The fee for attendance will be £3 3s., and those wishing to enrol are requested to make early written application to the Course Secretary, the Post-Graduate Committee in Medicine, Herford House, 188 Oxford Street, Paddington. Telephone: 31 0671. Telegraphic address: "Postgrad Sydney".

Course for D.A. Part II, and Final F.F.A.R.A.C.S.

An intensive full-time course suitable for candidates for the D.A. Part II and final F.F.A.R.A.C.S. examinations will be conducted in Sydney for two weeks from March 13 to 24, 1961, inclusive. Further particulars may be obtained on application to the Course Secretary, The Post-Graduate Committee in Medicine. The address and telephone number are given above.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR MARCH, 1961.

THE Melbourne Medical Post-Graduate Committee announces the following programme for March, 1961.

Pathology.

On March 6, classes will commence at the Pathology Department, University of Melbourne, for candidates for M.D. I, and Part II of M.S., M.G.O. and the diplomas. The course will consist of practical demonstrations followed by lectures from 1.30 to 3.30 p.m. on Mondays and Wednesdays, till the end of June. The fee is £21, and enrolments, on the Committee's form, should be received by February 20.

Physics.

On Thursday, March 16, an 18 weeks' course in physics for candidates for D.D.R. or D.C.R.A. in radiodiagnosis will begin. This will consist of lectures from 4.30 to 6.30 p.m. on Thursdays at the Commonwealth X-Ray and Radium Laboratory. The fee is £21, and enrolments should be sent to the Committee by March 2.

Psychiatry.

From Tuesday, March 14, the Australasian Association of Psychiatrists and the Mental Hygiene Authority will conduct a course in psychiatry. This will consist of lectures and discussions each Tuesday and Thursday until the end of August, commencing at 8 p.m. The course will be suitable for candidates for Part II D.P.M., and the fee of £15 15s. should be sent to the Melbourne Medical Post-Graduate Committee, together with the enrolment on the Committee's special form.

Radiodiagnosis.

In consultation with the College of Radiologists of Australasia, the Melbourne Medical Post-Graduate Committee will conduct a course in radiodiagnosis, consisting of about 24 lectures, from 4.30 p.m. on Mondays and Thursdays, commencing on May 1. A detailed time-table will be published later. The fee is £21, and should be sent to the Committee with completed enrolment form by April 14.

Microbiology.

A course in microbiology suitable for candidates for Part I M.D. and Part II M.S., M.G.O. and the diplomas, will commence on April 4 at the Bacteriology Department, University of Melbourne, and will continue at 2.15 p.m. for 20 weeks. The fee is £9 9s. Commencement will depend on a satisfactory number of enrolments. Those interested should communicate with the Post-Graduate Committee by March 21.

Symposium on Carcinoma of the Colon.

An invitation is extended to all medical practitioners to attend the symposium on carcinoma of the colon, to be held on Saturday, March 18, 1961, in the Public Lecture Theatre, Arts Building, University of Melbourne. The chairman of the symposium will be Mr. Charles Osborn, and the programme is as follows: 11 a.m., "Anatomy and Physiology", Mr. E. S. R. Hughes; 11.20 a.m., "Pathology", Dr. J. D. Hicks; 11.35 a.m., "Clinical Aspects, A", Dr. P. J.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 7, 1961.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	4(2)	..	..	..	..	..	..	5
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4(1)	9(7)	..	..	1	..	6	1	21
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	3(2)	..	..	..	..	..	..	3
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	108(52)	87(47)	21(4)	53(10)	10(10)	..	2	4	285
Lead Poisoning	..	..	3	..	..	..	..	..	3
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(1)	2(2)	..	..	..	..	..	..	5
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	6(3)	..	..	..	1	..	..	7
Puerperal Fever	..	1	1	..	..	..	..	..	2
Rubella	..	21(10)	..	..	16(15)	..	..	..	37
Salmonella Infection	..	..	..	1(1)	..	..	..	..	1
Scarlet Fever	4(2)	18(10)	3(1)	1	1(1)	..	..	..	27
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	..	..	..	..	..
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	20(14)	16(12)	21(7)	4(3)	1(1)	3(2)	..	..	65
Typhoid Fever	1(1)	..	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Parsons; 11.55 a.m., "Clinical Aspects, B", Mr. A. R. Kelly; 12.15 p.m., "Radiology", Dr. A. E. Piper; 1 p.m., film; 1.20 p.m., "Relationship to Ulcerative Colitis", Dr. I. R. Mackay; 1.35 p.m., "Relationship to Polyps", Mr. S. F. Reid; 1.50 p.m., "Treatment", Mr. Alwynne Rowlands; 2.10 p.m., "Results of Treatment", Dr. E. V. Keogh. After the presentation of papers, there will be an opportunity for discussion.

To assist in the arrangements, including catering, practitioners are requested to advise the Committee not later than February 24 of their intention to be present. Parking for cars will be available in the vicinity of the Arts Building, and during the symposium telephone messages may be relayed to the Lecture Theatre through FJ 5406. The symposium has been arranged by the Melbourne Medical Post-Graduate Committee in collaboration with the Anti-Cancer Council of Victoria.

Overseas Visitors.

The following lectures will be given at 8.15 p.m. in the Medical Society Hall.

On Thursday, March 2, Dr. Dudley Hart, physician and rheumatologist, of London, will speak on "Corticosteroid Therapy in Rheumatoid Arthritis: The Present Position". Dr. Hart's visit has been sponsored by Geigy Limited, and all practitioners are invited to attend the lecture, without fee.

On Tuesday, March 14, Professor W. F. Gaisford, pædiatrician, of Manchester, will speak on a subject to be announced. The fee for this lecture is 15s., but those who have paid an annual subscription to the Committee are invited to attend without further charge.

It is expected that Professor Erwin O. Strassman, obstetrician and gynæcologist, of Hueston, U.S.A., will visit Melbourne from March 8 to 15. Announcement of any lecture arranged will be made later.

Country Courses.

Details will be announced later of a week-end course to be conducted at Mooroopna.

Flinders Naval Depot.

On Wednesday, March 15, at 2.30 p.m., Dr. R. Zacharin will lecture at the Flinders Naval Depot on "Early Diagnosis of Genital Cancer". This lecture is given by arrangement with the Royal Australian Navy.

Address.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

Notice.

BRITISH MEDICAL ASSOCIATION, VICTORIAN BRANCH: ANNUAL CHURCH SERVICES.

ON Sunday, February 19, 1961, at 11 a.m., the twelfth annual special church services for the medical profession will be held at St. Paul's Cathedral and St. Patrick's Cathedral, Melbourne. The preacher at St. Paul's Cathedral will be the Reverend C. K. Hammond, M.A., Chaplain to the Alfred Hospital; and the preacher at St. Patrick's Cathedral will be the Reverend B. Lay, of St. John's Church, Mitcham. Members will enter the Cathedrals in procession, and are asked to assemble in the precincts fifteen minutes before the commencement of the services. It is desired that academic dress be worn, but this is not essential. All members (whether or not they are wearing academic dress) are asked to join the processions and sit together in a group. The Branch Council invites medical students to the services, and also to join the processions. Separate seating will be reserved for members' families.

VICTORIAN LAENNEC SOCIETY.

THE first meeting for 1961 of the Victorian Laennec Society will be held on Wednesday, February 22, 1961, at 5 p.m. in the lecture theatre, Royal Australasian College of Surgeons Building, Spring Street, Melbourne. Dr. D. Geraint James will be guest speaker, and he will present a paper entitled "Erythema Nodosum—A Review of 170 Cases". All members of the British Medical Association are invited to be present.

Deaths.

THE following death has been announced:

POATE.—Hugh Raymond Guy Poate, on January 27, 1961, at Sydney, N.S.W.

Diary for the Month.

FEBRUARY 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEBRUARY 17.—New South Wales Branch, B.M.A.: Ethics Committee.
FEBRUARY 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEBRUARY 23.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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